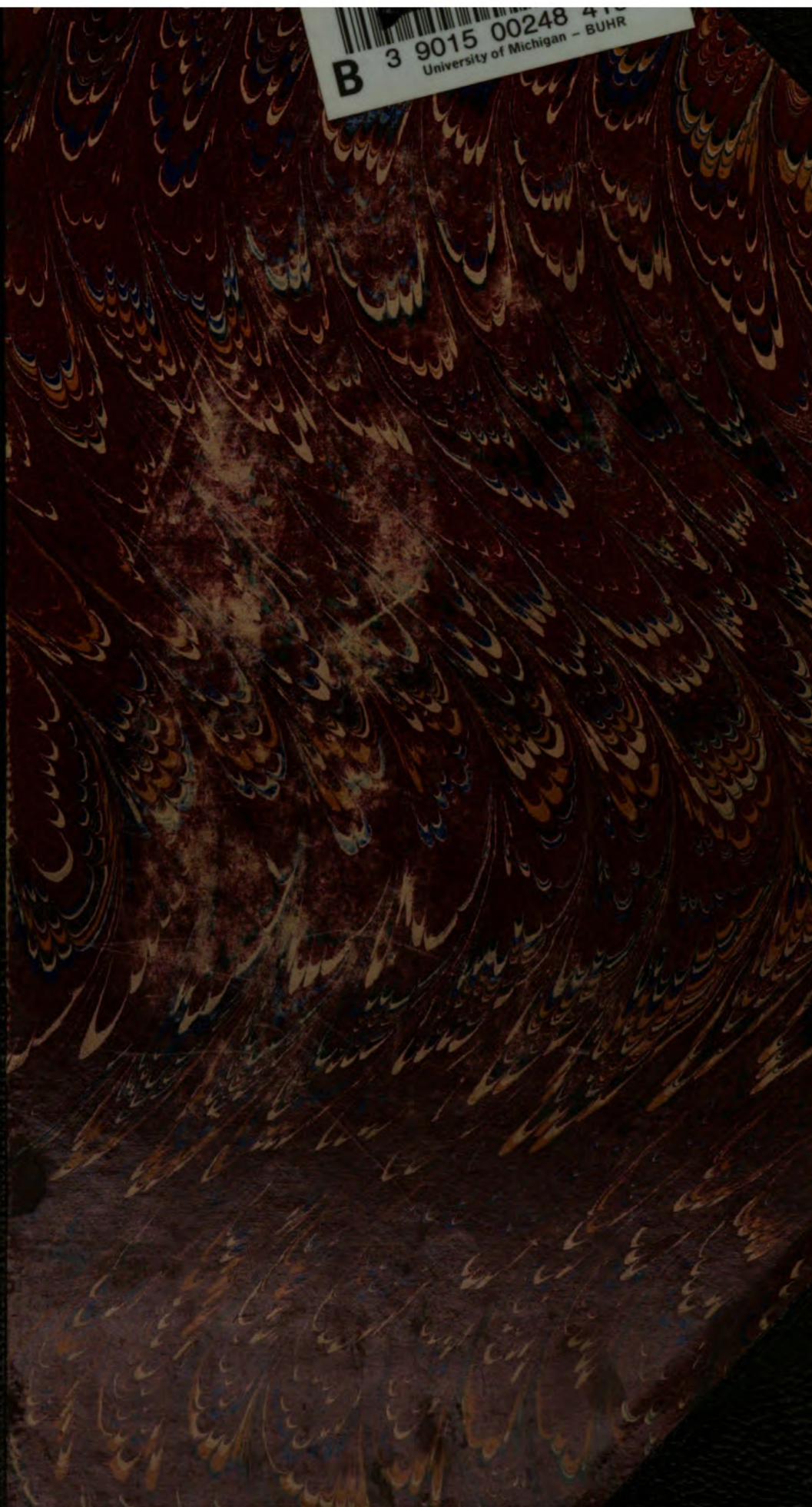
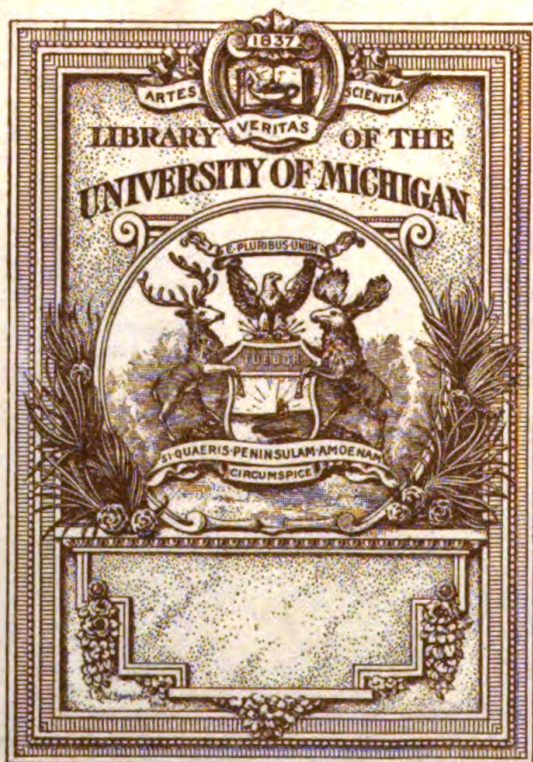


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REVIEW  
OF  
NEUROLOGY AND PSYCHIATRY

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# Review of Neurology and Psychiatry

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## PRELIMINARY STATEMENT.

THE object of the *Review of Neurology and Psychiatry* is to provide in English a journal similar to the various shorter reviews and Centralblätter on the same subjects which are published on the Continent in French, German and Italian, and which have proved of such assistance to those who are familiar with these languages. The Review is in no way intended to trench upon the field already occupied by the various larger British and American journals of nervous and mental disease.

The Review will appear in the first week of each month, and will extend, it is hoped, to forty-eight or sixty-four pages, according to the amount of matter available. Each number, it is hoped, will contain short original articles, or preliminary communications, abstracts of the most important original papers which have recently been published in every department of neurology and psychiatry, reviews of books, and a bibliography of all accessible recent articles, as well as occasional digests of recent progress in knowledge of special subjects.

The original articles or preliminary communications may extend from half a page to six or eight pages, and will be illustrated if the nature of the subject requires. The abstracts will, as far as possible, be made by those specially working at, or

pecially interested in, the particular subject to which they refer, and will be published as soon as possible after the appearance of the original paper. As a rule, an endeavour will be made to abstract all important papers that have appeared in the month previous to that in which the matter of the journal is made up—thus, the January number will consist mainly of references to work published in November, and so on. The bibliography will be found exhaustive and up to date, and will mention all books received by the Editor for review.

All communications regarding original articles should be sent to the Editor, and those regarding abstracts and reviews to the Assistant-Editor.

# Original Articles

## LOCAL PANATROPHY.

By Sir W. R. GOWERS, M.D., F.R.S.

A REMARKABLE case of local atrophic change was recently shown to the Clinical Society by Dr Harry Campbell. He was good enough to allow me to see it first, and I at once recognised its correspondence to one which I saw in private in 1885. I referred to it in my "Manual of Diseases of the Nervous System" (vol. i.), in the description of the varieties of spinal muscular atrophy,\* although I recognised that it has probably no relation to that disease.

My patient was a single woman aged 33 when I saw her. I read a brief account of the case at the meeting at which Dr Campbell's patient was shown, and a much fuller description of the present state of the patient will shortly be submitted to the Society by Dr Stanley Barnes, who has been able to make a careful examination of her condition, and finds that many fresh atrophic areas have developed. Yet, with one exception, she is hardly more conscious of the malady than she was seventeen years ago, and is now married.

I have suggested the name of Local Panatropy as the most fitting designation of this curious affection. In certain areas of the trunk, limbs, or face, which vary in diameter from that of a nut to that of an orange, or larger, there seems to be wasting of all the subcutaneous tissues down to the bones, with slight change also in the skin, which is there distinctly thinner and slightly discoloured. The aspect of the areas may be described as that of a subcutaneous excavation. They seem to be distributed quite irregularly, without apparent relation to the muscles or to the nerve distribution. Where the process is considerable the muscular tissue shares the wasting, but seems not entirely to disappear, and the electric irritability of that which remains is normal. Full details of the cases will be given in the Clinical Society's Transactions.

\* First ed. p. 365 ; third ed. p. 541 ; German ed. p. 487.



In my case there was no indication of implication of the bones. In Dr Campbell's case, besides such spots as I have described, there was a very similar condition of the whole of the right foot, in which the bones seemed likewise to have suffered some atrophy. This suggested to him a relation to facial hemiatrophy, and it is curious that I noted the analogy in the notes in my case-book made when I saw my patient (1885). Another case, yet to be fully described, which is under the care of Dr Savill, establishes the relation still more closely, since a similar condition is associated with definite facial hemiatrophy. Yet, in most cases of the latter, nothing of the kind has been recorded. In my patient, facial paralysis of the peripheral type has lately developed, but this is probably due to changes in the fibrous tissue about the nerve, since a small atrophic area exists between the ramus of the jaw and the ear.

Local panatrophie is certainly not a common disease, but it may often have been overlooked. The cutaneous change may have brought cases under the notice of dermatologists, but this is certainly the least important element in the condition. In Dr Campbell's case the skin is thin and discoloured, not only over the atrophic areas but also over some of the vertebral spines, as if from the effect of pressure ; but this cause cannot be assumed in the case of most of the areas, and especially in those in which the deep atrophy is greatest.

Until something is known of the actual pathological changes, it is difficult to form any hypothesis of its causation. Meanwhile the name I have suggested may serve to fix attention on the leading features, and to assist its recognition.

### **PSYCHIATRY IN GENERAL HOSPITALS.**

By Sir JOHN SIBBALD, M.D.

THE British Medical Schools are surely to be congratulated on the production of a journal in which the subjects of Neurology and Psychiatry are to be dealt with in combination. During the nineteenth century, and especially during the last fifty years, the student of scientific medicine, and also the practical physician, have recognised with steadily increasing conviction of its importance, the essential unity of the two subjects. It seems also

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to be with peculiar fitness that such a journal should be associated with the school of Cullen, who was the first to formally conjoin these subjects when in his Nosology he placed the *Vesaniæ* as one of the orders in the class which he instituted under the name of *Neuroses*.

With every forward step in our knowledge of the pathology of the nervous system, and with every new ray of light that has been thrown upon the nature of those diseases which are called mental, it has become more and more obvious that Cullen was right when he placed those affections among diseases of the nervous system. We know now that a psychic lesion is just as much dependent on defective action of the nervous structures as a sensory lesion or a motor lesion; and we see with greater and greater clearness every day that the curative treatment of each rests on the same fundamental principles. It does not seem to admit of question, therefore, that in order to obtain a comprehensive and true view of the nature either of those diseases dealt with at the present day under the heading of neurology or of those dealt with under the heading of psychiatry, we must study them together.

*Circumstances which have kept the study of Psychiatry apart from that of Neurology.*

If the subject required only to be looked at from the scientific point of view, it is probable that the idea of dissociating the diseases affecting the mind from the other neuroses would never have been entertained. But considerations of a social nature quite outside the scientific or medical aspect of the subject have led to patients suffering from mental disorder being kept separate from other patients and dealt with in an exceptional manner, and the object of this paper is to show that this separation has been effected more completely than is desirable even from the social point of view.

Some of the social reasons for the separation have ceased to exist. The superstitious ideas which less than a hundred years ago were associated with the occurrence of insanity have ceased to be entertained, or at least to have any practical influence. But the effect of mental disease in perverting, enfeebling, or destroying the ability of the patients to conduct themselves in the

manner necessary for their own welfare, and with due regard to the welfare of others, requires, both in their own interest and in the interest of society, that the majority of them should be placed under conditions in which their liberty is more or less restricted. In consequence of this a large number of the insane are treated, and must always in any civilised community be treated, as a class apart. It is recognised that the State has a special duty towards these persons, and with the view of fulfilling this duty the legislatures of this and other countries have, during the past century, made laws to secure that the insane shall be suitably provided for, and that they and society at large shall be adequately protected. The legislation in the various countries has inevitably been diversified in its details, being necessarily adapted in each case to the system of government, the social organisation, and the political genius of the country. The problem to be solved has presented itself in different shapes, and the solutions have differed accordingly. In this way the separation of the insane from the rest of the community, both as regards their legal position and the manner in which they are treated, has been made more complete in some countries than in others, and in no country has it been made more complete than in the United Kingdom. It is here that the largest proportion of the insane are provided for in special institutions, and that the largest number are under special supervision on account of their mental condition. The law in England especially pushes official control in regard to the treatment of the insane further than is necessary or even desirable, and it has the unfortunate effect of injuring the study of psychiatry by the practical exclusion of persons suffering from mental disorder from our general hospitals; for the requirements of the law must be regarded as one of the contributory causes of this exclusion.

It requires every person living away from home, either in an asylum or elsewhere, who can be certified to be of unsound mind, to be placed under the control of the lunacy authorities. No such person can be boarded anywhere for payment without the order of a magistrate, and this involves having been certified to be insane, being reported as such to the Board of Commissioners in Lunacy, and being subjected to periodical inspection. Even in charitable institutions and other places where no payment is received, the Commissioners may visit such patients and may

require periodical reports in regard to them. These requirements of the law, as Sir William Gowers\* admitted, though criticising them adversely in his recent address on "Lunacy and the Law," are intended "to prevent those of sound mind being deprived of personal liberty in an asylum or elsewhere, and to ensure proper treatment for those who are detained." Provisions of some sort in the direction in which these regulations tend are an essential part of any good lunacy law. They are referred to here to show how far-reaching is the influence of the English Lunacy Law, but specially to indicate how the law, by subjecting charitable institutions to control by the lunacy authorities if they receive patients certifiable as insane, tends to deter the governing boards from receiving such patients.

In so far as the law tends to exclude cases of certifiable insanity from the wards of general hospitals, its influence is in my opinion unfortunate; and it is still more open to objection in so far as it tends to prevent the governing authorities in these institutions from providing for the curative treatment of cases of illness affecting the mind which have not reached the stage when certificates of insanity can properly be given. Whatever be the cause, however, the fact is that in English hospitals few either of the early or the more fully developed cases are admitted, though of course they cannot be altogether excluded, patients, admitted on account of bodily ailments being often found after admission to manifest mental symptoms. It is right to state with reference to the exclusion of cases of mental disease from general hospitals, that the legal relations of such cases are not the only circumstances which bear in that direction. One of these is that people are apt to assume too readily that asylums are the proper places for every form of mental affection, and that there are no good reasons for admitting any such cases to general hospitals. And

\* An Address delivered before the Medico-Psychological Association, see *British Medical Journal*, November 22nd, 1902. Sir William objects strongly to the requirements referred to above as to patients who are received for payment. He regards them as far too wide in their application, and says that to comply with them would inflict serious injury on many patients. If a patient, whose best chance of recovery is temporary residence with someone who has to be paid, cannot be so dealt with because the case is one in which certification would be disastrous, it is obvious, he says, that either the law must be broken or the chance of recovery must be diminished. I agree with Sir William in his recommendation that the English law might with advantage be modified so as to bring it more into agreement with the less rigorous provisions of the law in Scotland.

another fact which should not be overlooked is, that even in the medical profession there are comparatively few who have given much consideration to the matter or whose interests have been touched by such exclusion. Such reasons as these have probably contributed to prevent wards for mental diseases being, as a rule, provided in Scottish hospitals, where the same legal obstacles as are met with in England do not exist.

*Reasons why cases of Mental Disease should be provided for in  
General Hospitals.*

Let us now for a moment look at some of the reasons which make the treatment of mental disease in general hospitals desirable; and in view of the special difficulties in England which relate to cases of confirmed insanity, I shall deal only with the admission of incipient or early cases.

Few words are necessary, I think, to show that it is in the interest of the patients that such provision should be made for them. Incipient cases are, as a rule, uncertifiable, and cannot therefore be admitted to asylums. For a large number of these cases among the poorer classes hospital treatment offers the best hope of cure; and in many cases incurable insanity is inevitable without it. If general hospitals do not give it, it cannot be obtained at all. But general hospitals are the best conceivable places for such cases. Wards for mental diseases do not require to be distinguishable from other medical wards, and residence in such wards does not entail the industrial and social injury that, as a matter of fact, follows residence in an asylum. Such wards have also the advantage over an asylum that a patient who goes there is saved from the mental shock which is often felt on entering an institution largely devoted to the care of the incurably insane. That this is no fanciful advantage I am sure; and I may mention, in support of the statement, that in asylums where there are separate buildings used as admission hospitals, it is common for patients to rejoice if they recover before they have been resident in any other part of the institution, and to be correspondingly depressed if they are transferred to other wards from the admission hospital. The most important reason of all, in the interest of the patients, is, that the patients the wards would receive being in the



early stage of their malady, are in the condition when there is the best chance of curing them. It is then that the restorative forces of the constitution may be enabled to regain control, that toxic processes may best be checked, and that exhausted brain-cells may be stayed in their progress towards disorganisation. Surely no more need be said to show that wards for mental diseases in general hospitals would greatly benefit the patients who might be received into them.

In the interest of the study of psychiatry such wards would be of use in any hospital, but of incalculable use in a general hospital connected with a medical school. The more that this aspect of the question is considered, the more do the advantages of such wards come into view. I do not wish to depreciate the opportunities for teaching and for study that are afforded in an asylum, nor do I wish to deny that there is much in regard to mental disease that can be shown in an asylum which will not be found in wards for cases only in the early stages of disease. But it is none the less true that it is in the early stages of mental disorder that the best opportunity is afforded for studying the causes of the disorder, the relation which the mental symptoms bear to the bodily condition and the way in which the mental symptoms are developed. All mental disorders originate in bodily disorders either functional or structural, and it is by a study of their relation to such disorders that we obtain a knowledge of their nature and can best learn how we may promote their cure. As Dr Maudsley says, "mental disorders are neither more nor less than nervous diseases in which the mental symptoms predominate, and their entire separation from other nervous diseases has been a sad hindrance to progress." The ideal scheme of study would be to study in association the cases in which those nervous diseases exhibit mental symptoms and the cases in which they are free from such symptoms. The more, therefore, that we can associate the treatment of so-called mental disease with allied bodily disease, the better for the study of psychiatry.

It must not be left out of view that not only do the early cases of mental disease in which hospital treatment would be beneficial suffer by their present exclusion from general hospitals, but that medical education suffers seriously also. It is mental disease in its initial stages that it is most important that the ordinary medical practitioner should become acquainted with, as

it is such cases that he will most frequently be called upon to deal with in his professional life. It will seldom if ever be of practical importance to him to know how a patient in an asylum ought to be treated. What it is of the utmost importance that he should know, is how to treat patients suffering from incipient mental disorder in their own homes, so that if possible relegation to an asylum may not be required. Owing to the absence of wards for mental diseases in general hospitals he is, as a rule, launched on his professional career without having seen a single case of the kind.

*Psychiatric Instruction in Germany.*

The provision made on the Continent and especially in Germany for clinical instruction in psychiatry has often been referred to in discussions which have recently taken place on the subject of this paper. It may therefore be of use if I append a short statement showing how this stands at present. As I have already explained, the problem presents itself under special conditions in each country, and therefore what is possible and expedient in Germany and elsewhere is not necessarily possible or desirable here.

I give in the following tabular statement a few details in regard to the provision that is made for such instruction in connection with the twenty German universities, two universities in Austria, and one in Denmark. The statement shows that in six cases the wards for mental diseases form part of general hospitals; in six cases the wards are close to the clinics for ordinary diseases; in six cases they are within a mile of the other clinics; in five cases they are at a greater distance; and in five cases information as to distance has not been obtained. In fourteen cases the wards receive only patients suffering from recent insanity; in ten cases both recent and chronic patients are received; and in four cases patients suffering from diseases of the nervous system without mental symptoms are also received. In ten of the places where only recent insanity is treated the average duration of the residence of patients is not over twelve weeks, and in four such places the average residence is longer. In the clinics for recent cases the medical staff including both visiting and resident members is an average of one to every twenty-five patients.

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INSTITUTIONS USED FOR CLINICAL TEACHING OF MENTAL DISEASES IN CONNECTION WITH GERMAN AND THREE OTHER UNIVERSITIES, A.D. 1900.

Names of Universities.	Wards form part of General Hospital.	Independent of General Hospital.			Classes of Patients provided for in the Wards.				Average sojourn of Patients.		Number of Physicians.		Date of Opening.	
		Close to other Clinics.	Within a mile of other Clinics.	Beyond a mile from other Clinics.	Recent Insanity.	Both Recent and Chronic.	Delirium.	Other Nervous Diseases.	Years.	Weeks.	Non-Resident.	Resident.	Of present Building.	Of regular Clinique.
Berlin . . .	1	1	...	...	65	...	26	60	...	3	4	3	1798	1865
Greifswald .	...	...	1	...	...	48	...	...	...	48	2	1	1834	1889
Kiel . . .	...	...	1	...	...	80	...	...	...	...	...	...	1900	1900
Göttingen .	...	...	1	...	...	345	...	...	2	66	4	3	1866	1866
Tübingen .	...	1	...	...	114	...	...	...	...	5	1	3	1894	1894
Rostock . .	...	...	...	1	...	200	...	...	45	1	2	2	1896	1896
Erlangen .	...	...	...	1	...	857	...	...	3	26	3	4	1846	...
Heidelberg .	...	...	1	...	80	...	...	...	...	21	2	2	1878	1878
Marburg .	...	...	...	1	...	300	...	...	1	3	2	2	1876	1876
Freiburg .	...	...	1	...	...	90	...	...	...	38	2	2	1887	1887
Breslau . .	1	...	...	...	215	...	...	...	...	10	1	2	1887	1877
Strassburg .	1	...	...	...	70	...	...	30	...	11	2	2	1886	1872
Würzburg .	...	1	...	...	60	...	...	...	...	12	2	2	1888	1833
Giessen . .	...	1	...	...	60	...	...	...	...	19	3	2	1896	1896
Jena . . .	...	...	...	1	180	...	...	...	...	20	2	2	1879	1848
Leipzig . .	...	1	...	...	130	...	...	25	...	12	4	4	1882	1882
Halle . . .	...	...	1	...	100	...	...	20	...	9	2	3	1891	1891
Bonn . . .	...	...	...	1	...	600	...	...	1	15	3	3	1882	1882
Königsberg .	1	...	...	...	30	...	10	...	...	7	2	1	1879	1892
Munich . .	...	...	...	...	...	530	...	...	2	12	2	5	1859	1861
Vienna, 1 .	...	...	...	...	...	800	...	...	...	44	3	5	1853	1853
" 2 . . .	1	...	...	...	130	...	...	...	...	4	5	3	1784	1875
Prague . .	...	...	...	1	158	...	...	...	...	13	1	3	...	...
Copenhagen	1	1	...	...	54	...	...	...	...	3	1	2	1888	1882

The position which Germany has made in providing for the clinical teaching of psychiatry is chiefly due to the impulse which Professor Griesinger gave to the movement during the three years in which, before his lamented death in 1868, he filled the chair of Nervous and Mental Diseases in the University of Berlin. I visited Berlin a few years before his appointment and saw the building attached to the Royal Charité Hospital in which the insane were located, and where restraint and seclusion and all the worst features of the old asylum treatment were rampant. And I remember with what delight I saw the same building in 1867 under Griesinger's *régime*. The change was like a miracle; and in recalling to mind what I saw there and

what I thought of it, I feel that I did not adequately realize how much that great man was in advance of his time; for there was little either in the treatment of the patients, or in the mode of conducting the teaching, that was in any way behind the best that is to be seen at the present day. When I came back to Scotland, being then the medical superintendent of one of the district asylums, I made an unsuccessful attempt to induce hospital authorities to introduce wards for mental diseases into the institutions under their charge, where psychiatry might be taught as it ought to be. I trust that the movement recently started by my friend, Dr John Macpherson, now one of the Commissioners in Lunacy, may not be so fruitless.

### **THE RELATIVE FREQUENCY OF DISSEMINATED SCLEROSIS IN THIS COUNTRY (SCOTLAND AND THE NORTH OF ENGLAND) AND IN AMERICA.**

By BYROM BRAMWELL, M.D., F.R.C.P.E.,  
Physitian to the Edinburgh Royal Infirmary, etc.

IN my experience, disseminated sclerosis is, comparatively speaking, a common disease in this country. It certainly seems to be very much more common here than it is in America. I have lately been making a special study of disseminated sclerosis, and have gone carefully through all my hospital and private casebooks. After excluding re-entries and re-admissions I find that (up to September 30th, 1902) I have notes, more or less complete, of 5825 cases of nervous disease (organic and functional), and that in these 5825 cases there were 100 cases of disseminated sclerosis; consequently 1 in every 58 nervous cases was a case of disseminated sclerosis. Of the 5825 cases, 3065 were private and 2760 hospital cases. In the 3065 private cases, there were 49 cases of disseminated sclerosis, or 1 in every 62 cases, and in the 2760 hospital cases there were 51 cases of disseminated sclerosis, or 1 in every 54 cases.

From this statement it appears that in my practice disseminated sclerosis is somewhat, though not much, more common in hospital than in private (consulting) practice.

I have lumped all the hospital cases (out-patients and in-

patients) together for the cases of nervous disease which are admitted to my wards in the Edinburgh Royal Infirmary are, for the most part, cases of organic disease—comparatively few cases of functional disturbance can, from the demands of the more serious cases, be accommodated. Further, the numbers of cases of disseminated sclerosis amongst my *Edinburgh* in-patients (37 in 727 cases of nervous disease, or 1 in 19)\* and out-patients (11 in 1857 cases of nervous disease, or 1 in 168) cannot be fairly compared. The much greater frequency of the disease amongst the in-patients is due to the fact that most of the cases of disseminated sclerosis which were first seen as out-patients were transferred to the wards and classified, not as out-patients, but as in-patients. The frequency of disseminated sclerosis amongst the different classes of patients who have come under my notice is seen in the following table:—

TABLE I.—*Showing the Frequency of Disseminated Sclerosis in Dr Byrom Bramwell's Hospital and Private Practice.*

Hospital or Private.	Cases of Nervous Disease.	Cases of D.S.	Proportion of D.S. to Total Nervous Cases.
Hospital—Newcastle In-patients .	176	3†	1 in 58
E.R.I. Out-patients .	1,857	11	1 in 168
E.R.I. In-patients .	727	37	1 in 19
Total Hospital Patients .	2,760	51	1 in 54
Private Patients . . . . .	3,065	49	1 in 62
Total Hospital and Private Patients . . . . .	5,825	100	1 in 58

And here I would point out that the proportion of cases of disseminated sclerosis to the total cases of nervous disease shown by these figures undoubtedly under-estimates the actual frequency

\* The proportion of 1 disseminated sclerosis to every 19 cases of nervous disease seems very high; but it is exceeded at the National Hospital for Paralysed and Epileptics, Queen Square, London. During the three years 1896, 1897 and 1898, there were treated in that hospital 2568 cases of nervous disease; of these, 159 were cases of disseminated sclerosis, giving a proportion of 1 disseminated sclerosis to every 14·8 cases of nervous disease.

† One of these three cases was diagnosed as primary lateral sclerosis, but was proved by post-mortem examination (20 years later) to be disseminated sclerosis.

of the disease, for in addition to these 100 cases which were definitely diagnosed as cases of disseminated sclerosis or which the future progress showed were cases of disseminated sclerosis, there were a considerable number of other cases in which a provisional diagnosis of disseminated sclerosis was made, but in which the diagnosis was less certain. These cases are not included in my figures, although some of them will undoubtedly turn out to be cases of disseminated sclerosis. Further, I have no doubt that amongst the cases entered in my notebooks as spastic paraplegia, lateral sclerosis, primary lateral sclerosis, ataxic paraplegia, hysterical paraplegia, hysterical paralysis, functional paralysis, etc., there were several cases of disseminated sclerosis.

The result of my observations then is to show that in this country (Scotland and the North of England) at least 1 out of every 54 cases of nervous disease met with in hospital practice, and at least 1 in every 62 cases of nervous disease met with in private practice, is a case of disseminated sclerosis.

Now contrast these figures with the American statistics. A discussion recently took place at the New York Neurological Society (February 4th, 1902) on the absolute and relative frequency of disseminated sclerosis.\* At that discussion a number of the leading American neurologists detailed their experience in hospital and in private practice. I have arranged their figures in the form of a table. From that table it will be seen that in 8000 private patients observed by Drs Dana, Hammond, and Sachs, there were 38 cases of disseminated sclerosis—in other words, 1 in every 210 cases of nervous disease in private practice was a case of disseminated sclerosis; while in 31,215 hospital cases observed by Drs Dana, Hammond, Allen Starr (Dr Goodhart), Onuf, Fisher, Collins, and Fraenkel, there were 141 cases of disseminated sclerosis—in other words, 1 in every 221 cases of nervous disease in hospital practice was a case of disseminated sclerosis. But, further, as Dr Graeme Hammond has pointed out to me, these statistics, *when taken as a whole*, are slightly fallacious, inasmuch as some of the cases of disseminated sclerosis included are duplicates—the same case of disseminated sclerosis not infrequently passing from one observer to another, and so being included as a new case by two or more different

\* Reported in *The Journal of Nervous and Mental Disease*, May 1902, p. 288.

observers. This does not, of course, in any way invalidate the statistics of any individual observer, but it gives a slightly exaggerated idea of the frequency of the disease in America when the statistics of several individual observers are added together.

TABLE II.—*Showing the Frequency of Disseminated Sclerosis in the Private and Hospital Practice of Several Leading Neurologists in America.*

Observer.	Private.			Hospital.		
	No. of Cases of Nervous Diseases	No. of Cases of D. S.	Proportion of D. S.	No. of Cases of Nervous Disease.	No. of Cases of D. S.	Proportion of D. S.
Dr C. L. Dana . . .	3,000	10	1 in 300	600	2§	1 in 300
Dr G. H. Hammond . .	3,000	15	1 in 200	7,000	32	1 in 218
Dr Goodhart (for Dr Allen Starr)	...	...	...	10,056	27*	1 in 372
Dr Sachs . . . . .	2,000	13	1 in 155	...	...	...
Dr B. Onuf . . . . .	...	...	...	say 550	8	1 in 68†
Dr E. D. Fisher . . .	...	...	...	2,451	8	1 in 306
Dr J. Collins (1898 to 1901) . . . . .	...	...	...	5,508	18	1 in 306
Dr J. Collins (1890 to 1897) . . . . .	...	...	...	4,000	28	1 in 150
Dr J. Fraenkel . . . .	...	...	...	1,050	18	1 in 58†
Total . . . . .	8,000	38	1 in 210	31,215	141	1 in 221†

My statistics, therefore, seem to show that, taking both private and hospital patients together, disseminated sclerosis is, revelatively to other forms of nervous disease, *at least three and a half times more frequent in this country than in America* (see Table III., in which the two sets of statistics are summarised).

§ "In both of these cases the diagnosis was questionable."

\* "In 6 of these 27 cases the diagnosis was doubtful, in other words, there was 1 undoubted case in 475."

† Note that if the figures of Dr Onuf and Dr Fraenkel were omitted, the proportion amongst the hospital patients would be 1 in 257 instead of 1 in 221.



TABLE III.—*Summary of the Two Sets of Statistics.*

Nationality, &c., of Patients.	No. of Cases of Nervous Disease.	No. of Cases of D.S.	Proportion of D.S.
American Private patients . . .	8,000	38	1 in 210
„ Hospital „ . . .	31,215	141	1 in 221
Total (P. & H.) patients .	39,215	179	1 in 219
Bramwell's Private patients . . .	3,065	49	1 in 62
„ Hospital „ . . .	2,760	51	1 in 54
Total (P. & H.) patients .	5,825	100	1 in 58

The difference is very remarkable. I cannot explain it. I do not think that it can be due, as Dana has suggested, to the fact that the circumstances and surroundings of the hospital class in America are better than the circumstances and surroundings of the hospital class in this country; for the difference is not confined to the hospital class; it applies equally to private practice—amongst my private patients the disease is three times more frequent than in the American hospital patients. Further, in my hospital practice, the disease is more common in the well-to-do (hospital) patients than in the very poor.

I am of course aware that in such a disease as disseminated sclerosis a comparison of the statistics of different physicians is apt to be fallacious, for one man may include cases which by another man would be excluded. It may, for example, be said that some of the cases which I diagnose as disseminated sclerosis, and have included in my statistics, would not be diagnosed as disseminated sclerosis by the American physicians. But although I admit that I now diagnose some cases as disseminated sclerosis which I would not have diagnosed as disseminated sclerosis ten years ago, I do not believe that it is possible to reconcile the difference in this way. In my series of 100 cases, 80 were so typical and characteristic that they would, I think, have undoubtedly been classed by every experienced neurologist as cases of disseminated sclerosis. In the remaining 20 cases, the symptoms were less typical; but I am satisfied that all of them should be classed as disseminated sclerosis. I have personally

analysed the notes and have carefully considered the diagnosis in every case. I have excluded a number of cases which were entered in the case books as cases of disseminated sclerosis or as probable cases of disseminated sclerosis.

But taking, for the sake of argument, the 80 typical cases alone—cases which every experienced observer would, I think, without doubt have regarded as cases of disseminated sclerosis—the proportion to the total number of cases of nervous disease in my private and hospital practice is 1 in 82. Whereas the proportion of cases of disseminated sclerosis, of all forms, met with in America by the physicians quoted above, was in private and hospital practice 1 in 219.

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## Abstracts

### ANATOMY.

#### **SUR L'ÉTAT DES PROLONGEMENTS PROTOPLASMIQUES DES**

- (1) **CELLULES NERVEUSES DE LA MOELLE ÉPINIÈRE CHEZ LES VERTÉBRÉS SUPÉRIEURS.** S. SOUKHANOFF and F. CZARNIECKI, *Névrose*, vol. iv. fasc. i. 1902, p. 77.

THE authors divide pieces of adult rabbit cord longitudinally into an anterior and posterior portion. After seven days in Golgi's fluid, the pieces are placed for two days in silver nitrate solution. In longitudinal sections the dendritic processes only of the largest cells are impregnated. Three types of cells are described.

1°. Bipolar cells with one dendrite from one pole and two from the other. These dendrites are irregularly varicose, and present gemmulæ in small number at wide intervals. These gemmulæ consist of a delicate pedicle ending in a globule.

2°. Fusiform cells with numerous dendrites. The dendrites are studded with gemmulæ close up to the cell-body. The gemmulæ vary in length, and some end in a delicate filament.

3°. Irregular cells lying in the white matter, with four dendrites. The portion of each dendrite lying in the white matter is devoid of gemmulæ and has an irregular contour. The gemmulæ appear first on the side of the dendrite turned towards the *white* matter. Short and long filamentous, pyriform, and rod-like gemmulæ occur.

The dendrites of the cells of the anterior cornua may be divided into three groups:—1°, Those of uniform thickness; 2°, those showing slight variations in thickness; 3°, moniliform dendrites. The second is the commonest form. Gemmulæ are very few in number.

The nerve-cells of the cord differ markedly from those of the cortex. The dendrites are more frequently varicose, and the gemmulæ are fewer in number and more variable in size and form. Peculiar offshoots intermediate in character, between gemmulæ and protoplasmic processes, occur and are totally absent in the cortex.

J. A. MURRAY.

**SUR LE RÉSEAU ENDOCÉLLULAIRE DE GOLGI DANS LES  
(2) ÉLÉMENTS NERVEUX DE L'ÉCORCE CÉRÉBRALE**

S. SOUKHANOFF, *Névraze*, vol. iv. fasc. i. 1902, p. 47.

**METHOD.**—Small pieces of rabbit cortex (3 weeks to 2½ months) as fresh as possible, are placed for five to seven days in Veratti's fluid.

Pot. bichrom.,	5 per cent.	1 part.
Pot. chloroplatinite,	1	" 1 "
Osmic acid,	1	" ½-¾ "

Use a large quantity and not too many pieces.

Change into cupric sulphate 5 per cent., 1 part; pot. bichrom. 5 per cent., 3 parts, the precipitate having been removed by filtration.

Allow to remain in this for twelve to fourteen hours, *i.e.* one-tenth of time in Veratti's fluid. Again change into 1 per cent. silver nitrate for twelve hours to two days. Dehydrate ten to fifteen minutes, first in 96 per cent. alcohol, then absolute, and fix on a cork with collodion. Clear sections after dehydration in guaiacol. Change into cedar oil, mount in immersion oil.

The network is perinuclear and does not reach the cell periphery.

The general arrangement corresponds with the cell-outline, sending branches into the protoplasmic processes, in some cases as many as two or three into one process. The network is composed of filaments of varying thickness, frequently moniliform and here and there flattened out in ribbon form. Soukhanoff's observations establish the endocellular character of the network firmly. As to its significance he inclines to regard it as a system of intracellular canaliculi. The general arrangement and the occurrence of ribbon-like fibrils suggest that the appearances are due to a partial impregnation of an ordinary protoplasmic foam-structure.

J. A. MURRAY.

**CONTRIBUTION À L'ÉTUDE DES APPENDICES SUR LE CORPS****(3) CELLULAIRE DES ÉLÉMENTS NERVEUX.** S. SOUKHANOFF,*Névrose*, vol iv. fasc. ii. 1902, p. 223.

SOUKHANOFF describes cells in the spinal cord showing gemmulae on the cell-body. In preparations from an old guinea-pig they were very numerous, completely covering the cell between the protoplasmic processes. They were also found singly in the cells of the posterior cornu in a man aged fifty-two, but are found much more frequently in the same situation in the cord of new-born children. The author decides against the view that these gemmulae may be artefacts, relying chiefly on their similarity to those covering the dendrites which have been demonstrated by the methylene blue method.

J. A. MURRAY.

**LO SVILUPPO DELLA CELLULA NERVOSA NEL MIDOLLO****(4) SPINALE DI POLLO.** O. FRAGNITO, *Ann. di Neurolo.*, 1902, f. 3.

THE author has previously made communications on this subject to the Congress held in Naples in 1899 and to the Congress recently held in Ancona.

In this paper he first draws attention to the importance of tracing the development of the embryo of the chick continuously from the beginning; and mentions as proof of the necessity for this, that if the stage of development, from the commencement of the seventh day to the end of the ninth day, is omitted, the most conspicuous part of the work of the grouping of the neuroblasts, of the formation of cell colonies, and of their transformation into nerve cells, will escape observation, and it will be concluded that a nerve cell is derived from a single neuroblast.

He then describes the appearances seen at the end of the third day of development, after briefly mentioning that he sees no grounds for supposing that the germinative cells are derived from the epithelial cells, but rather, that from the first they exist as two distinct types of cells.

By the end of the third day it is found that the epithelial cells have lost their typical structure and have been transformed into that fibrillary, lamellar tissue termed by His the "Neurospongium."

This fibrillated appearance is most marked towards the periphery, and as the neurospongium develops, its filaments become finer, its connections more numerous, and its network more regular.

Whether the neurospongium is formed entirely from the peripheral prolongations of the epithelial cells, or whether other



mesodermic elements take part, is a point which is not yet settled.

The author, however, asserts that at the time when the nerve cells are still in the stage of nuclei surrounded by a very thin layer of protoplasm, the neurospongium has already reached its full development, and is irrigated by blood-vessels and lymphatics.

It is in this spongioblastic network that the neuroblasts are found crowded around the central canal. In the embryo of three days they are round or oval nuclei with a dense, deeply staining, chromatic reticulum, with a well-marked contour, and surrounded by a very thin layer of protoplasm. This form is preserved until the beginning of the seventh day, the only changes which take place being a slight increase in size and a less intense staining reaction. Karyokinetic figures are very numerous.

The neuroblasts, at this stage are seen migrating peripherally, at first radically, and later in a direction which will lead them to their final destination. Some elements still preserve their primitive aspect.

At the end of the sixth day of incubation the elements have grouped themselves to a certain extent, but are not yet quite close to one another. At the beginning of the seventh day, in the anterior cornua of the cord, separate elements are no longer seen, but they are arranged in groups—cell colonies. The elements of which these colonies consist may be divided into two classes, primary neuroblasts, which go on to form the nuclei of the adult cell, and secondary neuroblasts, which are transformed into the various constituents of the adult cell protoplasm. In each colony several secondary neuroblasts are seen, and there may be more than one primary neuroblast, showing that in a colony it is possible for more than one adult cell to be formed. These elements are not the result of division of a mother cell, as some have supposed, because karyokinetic figures are seen in the nuclei only while they are crowded around the central canal. This is also proved by watching the subsequent course of development, in which the secondary neuroblasts are seen in various stages of transformation—with loss of contour, fusion of the elements, and absence of staining reaction with Thionin—around the more florid primary neuroblast, and at the origin of the protoplasmic processes.

The splendid plates which accompany this paper show very clearly the stages of migration, grouping and transformation of these elements.

Another reason against the derivation of these elements of the cell colonies from a single mother cell is, that by the fourth day many neuroblasts are present in the anterior cornua, but no karyokinetic figures are seen, and it is impossible to imagine that they should be quiescent from the fourth to the seventh

day, and, at the same time, show progressive rarefaction of the chromatic reticulum, and that they should then begin to multiply again.

Moreover the number of elements seen in the anterior cornua diminishes considerably during these days, and this although the volume of the cord has increased.

By the ninth day the elements differ very little in form from the adult nerve cell; it must not be understood, however, that all the nerve cells are developed contemporaneously.

The author concludes, therefore, that "one single neuroblast does not contain in itself all the elements necessary for the formation of a nerve cell, but that this originates by the fusion of a number, more or less large, of elements, proportional, perhaps, to their destined function."

He does not, however, consider that the secondary neuroblasts simply secrete the chromatin, but that the cytoplasm of the adult cell is formed by a transformation of all their parts, *i.e.* that the chromatin of the cell is provided by the chromatin of the secondary neuroblasts, and that the other parts of these neuroblasts are transformed into the other constituents of the cytoplasm.

With regard to the origin of the protoplasmic processes and the peripheral nerves, the author quotes the researches of several observers, who have established that the central and peripheral nerve fibres, and the cellular prolongations, which must be considered as the central extremities of these same fibres, are formed by the transformation of rows of cells.

It can be easily understood by connecting this idea with the idea that the body of the nerve cell is derived from a group of embryonal cells, how the embryonal equivalent of a cell process, or a nerve—a chain of cells—may, in part, apply itself to the peripheral layer of the secondary neuroblasts in a developing cell, and become incorporated with it, while its ends are continuous with those rows of cells from which the protoplasmic process or the peripheral nerve fibre, as the case may be, will be derived, and he figures one case in which this can be plainly seen. From these cells the nerve fibrils are formed: but the fibrils of the nerve cell differ from those of the peripheral nerve, in that, whilst the latter run an isolated course, the fibrils of the cell form a complicated anastomosis or network, which the author speaks of as the "specific reticulum of the nerve cell."

Without fully discussing the law established by Biervliet, that the chromatic substance is always deposited first in the peripheral part of the cell, the author says that he has frequently, and especially in the cells of the intervertebral ganglia, seen the primary neuroblast surrounded by a ring of secondary neuroblasts, which deposit the first chromatic substance immediately around

it, and then other layers are successively formed from the more peripheral rings of neuroblasts.

He then refers to the reticulum encircling the cell, described by Paladino, which consists of fine fibrils continuous with the interstitial tissue around, amongst which neuroglial nuclei are seen, and also mesoglia cells as described by Robertson. Later Paladino has traced neuroglial fibrils from this pericellular network into the cell body, some of which reach as far as the nucleus of the cell.

The author, however, suggests that it is more probable that these fibrils which are found in the cell body correspond to those fibrils of the neurospongium, which, with lymphatic and blood vessels, lay amongst the neuroblasts, when, at an early stage, they were grouped together to form cell colonies, than that they have penetrated from the pericellular network after the cell body has been developed.

Lastly, he referred to the presence of lymphatic vessels in the nerve cell, which reach the perinuclear space, as described by Holmgren and others.

Holmgren has said that these lymphatic vessels are furnished with a vessel wall, and are continuous with the pericellular lymphatic vessels; but the author, while he admits that some of the larger lymphatics may have a vessel wall and may be continuous with the pericellular vessels, is inclined to look on the greater number of them as corresponding to the original spaces between the secondary neuroblasts, which were the precursors of the nerve cell, and therefore without a true vessel wall.

Embryological, histological, and pathological data, then, compel us to distinguish two different tissues in the nerve cell: one, specific, derived from the transformation of the neuroblasts; the other, a supporting tissue, provided by the neurospongium, and by those blood-vessels and lymphatics which irrigate it.

R. G. Rows.

#### **A FURTHER NOTE UPON THE PRE-PYRAMIDAL TRACT**

(5) (MONAKOW'S BUNDLE). E. H. FRASER, M.D., *Jour. Physiol.*, Sept. 1902, p. 366.

EXPERIMENTING upon monkeys and cats, the author has made isolated lesions of the mesencephalon by means of a protected galvano-caustic point. He finds it convenient to use the upper surface of the bony tentorium as a guide for his instrument in making the lesions. The areas destroyed were strictly unilateral and were situated respectively in the dorsal part of the upper pons; deeply seated in the lateral part of the pons; in the anterior

colliculus mesio-laterally; in the posterior colliculus; in the thalamus postero-mesially; and in the fasciculus lateralis proprius medullæ.

The tissues were examined by the Marchi method, and the nerve cells of the red nuclei were also examined. The position of the lesions of the resulting degenerations are well figured.

The following conclusions are arrived at:—

The fibres of Monakow's bundle have their origin in the cells of the opposite red nucleus and none of the fibres arise in the thalamus nor in the superior colliculus. These fibres taking the course usually described in the brain stem, occupy a definite "pre-pyramidal" position in the lateral column of the cord in the cat, while in the monkey they are more or less scattered among the fibres of the lateral pyramidal tract. In both cat and monkey the fibres end in the posterior cells of the ventral horn of the cervical and lumbar enlargements, but no fibres enter the grey matter in the thoracic region.

Monakow's bundle contains no ascending fibres such as have been figured by Probst and others.

A very important fact brought forward in this article is the absence of any collaterals from the rubro-spinal tract passing to the grey matter of the thoracic cord.

The writer's terminology is unfortunate in that the term "pre-pyramidal" (used first, and it appears, only by Thomas for a degenerated area in front of the lateral pyramidal tract, not definitely traced upwards) cannot be said to apply to the position of the rubro-spinal tract in the spinal cord of man and of many other animals. The terms rubro-spinal tract and Monakow's bundle have been so long in use as not to warrant the introduction of a new and un-descriptive name into the already confused and redundant terminology of nervous anatomy. The same exception may be taken to the revival of the term "anterior columnar tract" (Boyce), for those fibres passing from the dorsal grey matter of the superior colliculus *via* Meynert's decussation to the ventral column of the spinal cord, to which the term ventral tecto-spinal tract (ventral longitudinal bundle) has been applied.

The writer is in error in stating that ascending fibres were described by Collier and Buzzard in the position of the rubro-spinal tract. These writers described descending fibres only.

J. S. COLLIER.

**UEBER DIE BEZIEHUNGEN DES UNTEREN LÄNGSBÜNDELS**

(6) **ZUR SCHLEIFE UND ÜBER EIN NEUES MOTORISCHES STABKRANZSYSTEM.** Von H. SCHÜTZ, *Neurolog. Centralbl.*, Oct. 1902, p. 885.

In two cases of almost complete absence of the temporal and occipital lobes, the author found the fasciculus longitudinalis inferior of normal size, and passing from the occipital, not to the temporal lobe, but to the mid-brain. He undertook further investigations with the embryological method, and came to the conclusion that it contains two sorts of fibres: (1) some which come from an area in which the optic tracts break up, lateral to the corpus geniculatum laterale; and (2) others which come directly from the fillet, and besides taking part in the formation of this bundle, pass to the first temporal gyrus, to the central convolutions, and to the nucleus amygdalus and cornu Ammonis.

Distalwards these fibres end in the substantia nigra, or form that lateral part of the fillet, identical in position with Schlesinger's lateral pontine bundle, and Hoche's lateral fillet tract. These, he believes, without furnishing independent proof, end in the nuclei of the motor cranial and spinal nerves, and so represent a *primary motor tract* from the olfactory, optic, acoustic and sensomotor centres. He regards it as a cortico-motor reflex system perhaps actively functional in hemiplegics and new-born children, and it may be directly responsible for epileptic seizures with *sense auræ*, in which the pyramidal tracts take no part.

Thus he agrees with Flechsig that it is a projection-system, and not, as usually believed, an association-tract between the occipital and temporal lobes.

GORDON HOLMES.

**DEGENERATIONS FOLLOWING LESIONS OF THE RETINA IN**

(7) **MONKEYS.** HERBERT J. PARSONS, *Brain*, Autumn, 1902, p. 257.

In this experimental research a lesion of a monkey's retina was made by means of a Graefe cataract knife. Observations were made on six monkeys. The animals were killed from a fortnight to three weeks after the retina had been injured. The resulting degeneration of the nerve fibres was traced in the optic nerves, chiasma, and optic tracts.

The parts were hardened in formol, then washed with water and transferred to Busch's solution. They were embedded in celloidin.

Parsons finds that the degeneration in the optic nerve on the side of the lesion corresponds, upon the whole, with that found



by others in rabbits. The different parts of the optic nerve correspond in general to the similarly situated parts of the retina. He finds, however, "that in the posterior part of the nerve as it approaches the chiasma, the nasal fibres tend towards the ventral side, and the temporal fibres tend towards the dorsal side." The course of the papillo-macular bundle in the optic nerve of man as determined by pathological investigation of cases of toxic amblyopia would appear to correspond to what is found in the monkey by experiment. The author confirms the observation that signs of degeneration are found in the optic nerve of the uninjured side; he believes that there is a true degeneration of inter-retinal fibres.

Both optic tracts were found degenerated in every case. More fibres appeared to be degenerated in the tracts than in the nerve. This is attributed by the author to sub-division of the optic fibres.

C. H. USHER.

**SULLA PRESENZA DI NEVROGLIA NELLA STRUTTURA DEI**

(8) **PLESSI COROIDEI.** G. CATOLÀ, *Riv. di patolog. nerv. e ment.*, vol. vii. f. 9, 1902.

THE author was led to make his investigation by the observation of a glioma of the choroid plexuses of the fourth ventricle. He has employed a modification of Weigert's method for the neuroglia, consisting in the application of the mordant to celloidin sections of the formalin-hardened tissues instead of to the pieces. He finds that the human choroid plexuses contain a delicate layer of undulating neuroglia fibres subjacent to the epithelium. Most of the fibres run longitudinally with respect to the long axis of the villi; others are arranged transversely and some encircle the vessels. He distinguishes three layers composing these organs, namely, the epithelial layer, the layer of neuroglia, and the connective-vascular or pial layer.

W. FORD ROBERTSON.

**NOTE ON FETAL MUSCLE SPINDLES.** By LAURA FORSTER,

(9) *Jour. Physiol.*, vol. xxviii. No. 3, 1902, p. 201.

MISS LAURA FORSTER, whose former work on the muscle spindle is well known, describes in the present paper the histological appearance of the muscle spindle in human embryos of the fourth, fifth, and sixth months.

The points in which the muscle spindle of the fourth month foetus differs from those in the adult are—

- (1) There is no lymph space.
- (2) The sheath of the spindle is very thin and difficult to detect.

- (3) The intra-fusal muscle fibres are on an average equal in size to the extra-fusal muscle fibres, whereas in the adult the intra-fusal fibres are smaller than the extra-fusal.

The intra-fusal muscle fibres probably develop earlier than the extra-fusal fibres, since a "hollow" appearance which is characteristic of developing muscle is present in the extra-fusal muscle fibres, whereas the intra-fusal fibres do not present this appearance, and are stained throughout.

At the fifth month of foetal life the lymph space begins to develop and the sheath becomes thicker, and by the sixth month the extra-fusal muscle fibres are frequently larger than the intra-fusal fibres.

These observations appear to show that the development of the muscle spindle progresses with the growth of the foetus, thus confirming the view that the spindle is of physiological importance for the fully developed muscle, and is not the vestige of an embryonic structure.

F. E. BATTEN.

**DIE VARIETÄTEN IN DER ANLAGE DER FISSURA CAL-**  
 (10) **CARINA UND DIE FISSURA RETROCALCARINA.** Von  
 v. MONAKOW, *Arch. f. Psychiat.*, Bd. 36, H. I.

AFTER pointing out the variability that exists in the arrangement of the cerebral fissures, von Monakow, from an examination of eighty hemispheres of different ages, describes four types of arrangement in which the calcarine fissure may be present, as a single stem, communicating with the parieto-occipital fissure, or subdivided into parts. The retrocalcarine fissure is a relatively constant deep fissure springing from the posterior part of the calcarine, whose margins show the same histological characters as the calcarine, and belong to the visual centre. He concludes that the convolutions of the parieto-occipital region are of no importance as lines of demarcation between cortical sensory areas, and that, as the variations can be seen at the time when the fissures are first laid down, it is unlikely that they can be acquired by education and experience.

DAVID WATERSTON.

**PHYSIOLOGY.**

**RECHERCHES EXPÉRIMENTALES SUR L'INNERVATION**  
 (11) **MOTRICITÉ DU LARYNX CHEZ LE LAPIN.** By FRITZ DE  
 BEULE, *Névrose*, vol. iv. f. 2, Oct. 15th, 1902, p. 163.

NOTWITHSTANDING all the work that has been done upon the motor innervation of the larynx, the subject apparently still presents itself as a field for further experimental investigation.

The paper now under review is the most recent addition to an already long list of contributions dealing with the somewhat vexed question of the laryngeal nerve supply. A study of this subject naturally resolves itself into three main divisions:—1. What muscles of the larynx does each of the laryngeal nerves supply? 2. Do both the vagus and the spinal accessory nerves furnish motor filaments, and if so, what part does each play in the muscular innervation? 3. Which is the medullary centre for the laryngeal nerves? It is to an elucidation of these three points that Fritz de Beule turns his attention. It would be outside the scope of a short review of this kind to follow the author through the historical aspect of the subject on these three questions, notwithstanding that this forms a valuable addition to his paper. It will be necessary, however, to refer briefly to the views held by some of the earlier workers in this field.

In dealing first with the individual muscles in which the nerves terminate, it is necessary to mention the classical work of Longet (1842), and the more recent experiments of Exner. The former held that all the muscles, with the exception of the crico-thyroid, derived their motor supply from the recurrent laryngeal nerve, while the external laryngeal branch of the superior laryngeal nerve innervated the crico-thyroid muscle. Further, that these nerves were only distributed to the muscles of the corresponding half of the larynx. The majority of physiologists have accepted this view. Exner, however, supported by a minority, endeavours to show that the arrangement is not such a simple one. To state his views very briefly and generally, we find that each crico-thyroid muscle is supplied partly by the superior laryngeal and in part by the middle laryngeal nerve of its own side. The other muscles may be innervated not only by both the superior and inferior laryngeal nerves of the corresponding side, but also by the same nerves upon the opposite side of the body. The middle laryngeal nerve described by Exner as existing in rabbits is a fine branch derived from the pharyngeal filaments of the vagus. The conclusions arrived at by De Beule as the result of a number of carefully performed experiments are these:—

1. The theory of Exner and his supporters, which advances the view that the muscles of the larynx have a double nerve supply, and that their supply is also derived from the nerves of both sides, is not in accord with facts.
2. The old and classical scheme of Longet should be preserved in its integrity, with the addition, perhaps, of the part played by the "middle laryngeal nerve."
3. The superior and middle laryngeal nerves each innervate parts of the crico-thyroid muscle.
4. The recurrent laryngeal nerve alone supplies all the other muscles, and this innervation is unilateral, with the exception, perhaps, of the arytenoideus muscle.

If we turn now to the second part of the research, namely, the relative part played by the vagus and the spinal accessory in the motor innervation, here we find that there has been very considerable investigation, and much difference of opinion has resulted. According to the author, these differences have arisen in part, at anyrate, from the methods employed in the research. Those who have worked on a physiological basis, such as Grosmann, Grabower, and Onodi, have eliminated the spinal accessory nerve from any participation in the motor supply. While those who have worked along anatomical lines, *e.g.* van Gehuchten and Bochenek, have concluded that it innervates, in whole or in part, the laryngeal muscles. For these reasons De Beule has employed both methods, the one controlling the other. It is also important to know, in relation to this part of the subject, which of the bulbar roots belong to the spinal accessory nerve and which to the pneumogastric. We need not follow the author through this part of his subject, but will content ourselves with stating the results at which he has arrived:—

1. After tearing across the spinal accessory nerve within the vertebral canal, Wallerian degeneration shows itself only in the corresponding inferior laryngeal nerve, but involving only a certain number of the nerve fibres. The superior laryngeal, and some fibres of the recurrent nerve which remain unaffected, must be tributaries of the vagus.

2. Isolated rupture of the bulbar roots of the vagus immediately arrests respiratory movement of the corresponding vocal cord, and excitation of the common vagus trunk in those animals which had survived three weeks after intracranial rupture of the spinal accessory produces movements at the glottis.

The question then arises as to the relative part played by each of these nerves. To this aspect of the subject De Beule also devotes himself, and concludes with the statement that while the spinal accessory nerve innervates the thyreo-arytenoideus externus, the vagus gives motor filaments to all the other laryngeal muscles.

Finally, we must turn to the third point, namely, which is the bulbar centre for the laryngeal nerves, and from his experiments the author makes the following deductions, *viz.*, that the dorsal vago-spinal accessory nucleus is the nucleus which innervates the muscles of the larynx. The cells which give origin to the motor fibres of the superior laryngeal nerve are found in the lower two-thirds of that part of the dorsal nucleus which belongs to the vagus. As to the cells of origin of the recurrent laryngeal nerve, they occupy in part the lower two-thirds of a portion of the dorsal nucleus belonging to the vagus, and in part the upper three-fourths of the spinal accessory portion of the same nucleus.

A. LOGAN TURNER.

**PATHOLOGY.**

**SOPRA ALCUNE PARTICOLARITÀ DI STRUTTURA DELLE  
(12) CELLULE DEI GANGLI SPINALI DELL' UOMO.**  
M. SCIUTI, *Ann. di Neurolo.*, 1902, f. 3.

FOR his work Sciuti has used the posterior root ganglia from two cases of tabes dorsalis, and in addition to finding degenerative changes he has found and described two networks in the cell.

The first is on the cell, or pericellular, and consists of large meshes formed by distinct cords running along one diameter of the cell and connected by finer threads. At the points of junction of the rami there are deeply stained nodal points. This reticulum passes into the surrounding tissues and may help to form two adjacent cell reticula. The fibres are often lost in the fibrillar cell capsule. When the cell colours homogeneously, then the reticulum is very evident; but where degeneration or pigment is present in the cell, the network is not perceptible.

The second or superficial network is situated at the periphery of the cell, and beneath the pericellular one, and is formed of finer filaments. These stretch all over the cell, forming connections with the first reticulum and also with the cell capsule. They are also connected with the prolongations of the small cells which stand inside the cell capsule.

Sciuti's reticulum has no connection with the capillaries or with the lymphatics, but is, in his opinion, of neuroglial origin and identical with the reticula of Golgi, Donaggio, Cajal and others. It is connected with the pericellular reticulum, with fibrils from the cell capsule, and with filaments from the special endo-capsular cells.

DAVID ORR.

**SUL SIGNIFICATO DELLE MODIFICAZIONI PATOLOGICHE  
(13) DELLA PARTE CROMATICA DELLE CELLULE NERVOSE.**  
E. LUGARO, *Riv. Speriment. di Freniatria*, 1902, f. i. p. 981.

THE author recalls the fact that Marinesco was the first to attempt to give a classification, from the standpoint of pathogenesis, of the morbid alterations that occur in nerve-cells. He distinguished secondary alterations due to indirect traumatism, from primary alterations the result of direct actions (mostly toxic), or of nutritive disturbances. The former, as observed after section of the nervous prolongation of the cell, consist in central chromatolysis with displacement of the nucleus to the periphery; the latter, whilst they may involve the chromatic substance of the whole cell diffusely, are most marked in the more peripheral zone, and are

therefore represented, at least temporarily, by a peripheral chromatolysis without displacement of the nucleus.

Although this teaching has been widely accepted, certain facts have been observed which tend to show that the two forms of alteration are not always so distinct from each other as has been supposed. In 1897, R. A. Fleming recorded the observation that after section of peripheral nerves in the rabbit, many of the cells of the posterior root ganglia, instead of exhibiting central chromatolysis with displacement of the nucleus, show a nucleus remaining central, condensation of the chromatic substance around it, and a peripheral zone devoid of chromatic substance. In the following year, Cox confirmed this observation, and concluded that in the spinal ganglion of the rabbit there is a special type of cell which undergoes this form of alteration upon section of the corresponding peripheral nerve. Further, Marinesco has made the observation, which has been fully confirmed by others, that if the nervous prolongation suffers a very severe mutilation, as from tearing out of the nerve-root, the cell may present a diffuse change consisting in almost complete dissolution of its chromatic substance, without displacement of the nucleus, and then undergo atrophy or degeneration, without presenting any of the typical characters of secondary degeneration.

With the view of shedding further light upon the question, Lugaro has made an extensive investigation of the effects of section of nerves on the cells of the sensory ganglia in various species of animals. He gives a summary of the results of these experimental observations. He concludes that it is probable that the type of reaction in which there is peripheral chromatolysis represents a less intense form than that in which there is central chromatolysis and displacement of the nucleus, and that therefore it tends to occur in certain types of cell which react less than others, and especially in the initial stage of the reactive period and towards the end of the reparative stage. He endorses the opinion of van Biervliet and van Gehuchten that the classical type of reaction of the cell to section of its nervous prolongation represents a rejuvenescence of the cell in association with regenerative activity in the injured nerve-fibre. In support of this view, van Biervliet has pointed out the resemblance that the reacting cell has to an embryonic nerve-cell. Lugaro adds the observation that types of cells corresponding to the phases of reaction and repair are found in certain stages of phylogenetic development, and that the slighter form of reaction—with peripheral chromatolysis, central nucleus and perinuclear aggregations of chromatic particles—is normal in some of the nerve-cells of the lower vertebrates.

On the ground of these facts Lugaro thinks that we should abandon the distinction between primary and secondary lesions,



founded upon changes in the chromatic part of the cell, and that we should speak rather of reactive and degenerative lesions (which, however, may co-exist in various proportions), the first of which is evidence of the reaction of a cell that is attempting to repair injuries directly or indirectly sustained, the second of the more or less grave damage of the cell by external agents.

He refers to the remarkable fact, which was first observed by himself, that whilst the nerve-cells of the posterior root ganglia readily react to injury to their peripheral prolongations, they do not react at all to section of their central prolongations. He previously explained this phenomenon as indicating that in maintaining the trophic condition of these cells the reception of stimuli is more important than the discharge of these stimuli. He now thinks that this hypothesis ought to be abandoned, as it is no longer admissible that the reactive modifications of cells depend upon the non-reception of stimuli or the suppression of discharge. It is only the cellular mutilation that is of importance. The reactive phase is dependent upon regenerative efforts on the part of the peripheral fibre. He rejects the view that the typical picture of *réaction à distance* can be produced by a lesion of other neurons which carry stimuli to those showing the change. He contrasts the very limited power of repair possessed by the central nervous tissues with the considerable reparative power of the peripheral nerves, and accounts for the difference on evolutionary grounds. The sensory neurons, in common with other peripheral neurons, have great power of repairing peripheral mutilations. Therefore they react when their peripheral branch is injured, but exhibit no reaction to injury to the central branch.

W. FORD ROBERTSON.

**SULL' ATROFIA SECONDARIA INDIRETTA DEGLI ELE-**  
**(14) MENTI NERVOSI.** E. TANZI, *Riv. di Patol. nerv. e ment.*,  
 1902, f. 8.

THE author in this work has taken up the study of the changes in the nervous elements of centres from which the sensory stimulus has been removed through destruction of the corresponding organ of sense. For his purpose he has chosen to study the visual centres on account of their situation and connections.

The following experiments were performed:—(1) *Exenteratio bulbi* was done on one side in six rabbits immediately after birth, and the animals were killed at the end of six months. (2) The same operation was performed on both sides in two rabbits. These were also killed at the end of six months. (3) In two dogs both eyes were enucleated on the day of birth, and



the animals were killed at the end of one year. The series was completed by the study of the brain of a dog blind from birth. This animal was killed when one year old.

The methods employed by Tanzi for his work have been those of Weigert, Nissl and Cox. In the rabbits in the region of the anterior bigeminal eminence he has noted the following changes on the injured side by Weigert's method: complete disappearance of the fibres in the superficial layer, reduction in thickness of the superficial grey layer and a thinning of the optic layer. Beneath this there was nothing abnormal to be seen. By Nissl's method the marginal cells were seen to be completely gone; and in the first grey layer, and in the vicinity of the optic layer, the cells were reduced in size but retained their structural characters. In the external geniculate body, the dorsal portion showed marked atrophy on the side opposite to the lesion. This was a little more marked in the animals operated on on both sides. The "stratum zonale" was greatly atrophied, but the cells were only reduced in size, retaining their morphological characters. In the cerebral cortex there was nothing abnormal to be noted, even when the animal had been totally blinded.

The brains of the dogs on which bilateral enucleation was performed showed slight changes to the naked eye, consisting in narrowing of the gyri, splenalis, suprasplenialis, and post-splenialis. In the bigeminal eminence the medullated fibres of the superficial layer, the underlying grey layer, and the optic layer were reduced in numbers. The nerve cells were diminished in size and more closely packed together than normal. The external geniculate body was smaller than normal, and had lost many fibres. In the cerebral cortex the above-mentioned convolutions were diminished in thickness, and Weigert's method revealed the fact they had lost many fibres. In the line of Gennari the fibres were also thinner and scarcer than in the normal brain. By Nissl's method the cells of all the layers were smaller, but especially in the superficial layers. The cerebellar aspect of the gyri, splenalis, post and suprasplenialis showed the most change. In the tissues of the anophthalmic dog the same changes were found as in the immediately preceding experiments.

*Conclusions.* — From his research the author concludes that lesions of the nervous system in new-born animals can cause complete degeneration in the neurons directly affected, but those which are simply connected with them and not directly injured undergo only a process of atrophy, never one of destruction. Thus in the writer's work the primary optic paths were destroyed and the secondary ones atrophied, but the cells preserved their shape and intimate structure. In the nuclei where the

primary paths terminate, the nerve terminations of the primary neurons were atrophied. The atrophy is an interstitial one, and affects the dendritic terminations of the secondary neurons also. The interstitial grey substance is thus diminished, and therefore the cells become placed closer together. In the visual area the atrophy of the neurons is of the same type as that in the neurons of the second order, but the fibre atrophy and consequent approximation of the cells is less, as there is no destruction of the intercellular terminations of the neurons of the antecedent order. Tanzi does not accept the view that this want of cortical atrophy is due to a substitution of function, nor does he believe that a neuron must degenerate if deprived of its customary stimulus. He believes that the fundamental trophism of the cell is sufficient to preserve its integrity. Even when the functional stimulus is suppressed before the cell is completely developed, the cellular trophism can carry the cell to complete development. In comparing the results obtained in the higher animals and in man with his own, he shows how in the more highly-developed brains the cortex is more sensible to suppression of stimuli, and thus in man and in the dog cortical changes are present which are absent in the rabbit. The dynamic trophism for the exercise of special function is acquired by the cell slowly, and therefore is more marked in the higher and more evolved species.

DAVID ORR.

**ON CHANGES IN THE CENTRAL NERVOUS SYSTEM IN  
(15) THE NEURITIC DISORDERS OF CHRONIC ALCOHOLISM.**

By SYDNEY JOHN COLE, *Brain*, Autumn, 1902, p. 326.

IN this paper there are the records of three cases of alcoholic neuritis. The *first*, a woman aged 32, had a history of eight years' alcoholism with several attacks of delirium tremens. There was multiple neuritis involving more or less completely nearly all the nerves of the body. Delusions, illusions, loss of memory and mental confusion were also prominent symptoms. The neuritis had a duration of six weeks.

In the *second* case, a woman aged 49, there had been eight months' excessive drinking, and probably several attacks of delirium tremens. She was under observation for five weeks, and during that time her condition was one of almost constant delirium. The legs were paralysed, but the other muscles of the body seem to have been little affected.

The *third* case (aged 45), also a woman, had been drinking for several years. There was marked mental confusion, and multiple neuritis affecting the nerves in both upper and lower

limbs, though some nerves more than others. The duration of the neuritis is not stated; it is, however, said to be "chronic." The mental disorder lasted about a fortnight, though the patient had a similar attack three years before.

A *post-mortem* was made in each case, and the results are as follows:—

In case 1 there was marked parenchymatous degeneration (Marchi's stain) of all the nerves examined. In cases 2 and 3 the tibials showed advanced interstitial changes, but the other nerves were practically normal. In all the cases the pyramidal tracts (from internal capsule to sacral region) showed degenerated fibres, these being most abundant in case 2, less so in case 1, and least of all in case 3. The fronto-thalamic fibres were also affected in each. In the posterior columns the degeneration was most marked in case 1, and least in case 3. In cases 1 and 3 it was entirely confined to exogenous fibres, but in case 2 the endogenous fibres were also involved. The direct cerebellar tract showed marked degeneration in case 2, slight in case 3, and not at all in case 1. The posterior roots, external to the cord, seem to have been but little affected. The cortical cells showed marked chromatolysis in the three cases. This was also the condition of the ganglion cells in the nuclei of the pons and medulla, and likewise of the anterior horn cells, though in case 1 these last were much more affected than in case 3 and rather more than in case 2. The spinal ganglion cells showed definite degeneration in case 1, slight in case 2; in case 3 they were not examined. The cells of Clarke's column were normal in case 1, degenerated in case 2, and slightly degenerated in case 3. In case 2 the walls of the vessels were thickened and atheromatous, but in the other two cases they showed little change.

The paper discusses the above appearances in detail. It concludes that the peripheral neuritis is not secondary to the cell lesion, neither is the degeneration of the cell secondary to the degeneration in the nerve, for the two are just different expressions of the same neurone degeneration. Alcoholic neuritis, too, is not peripheral only, for a similar degeneration is found in neurones entirely central; the affection is therefore poly-neuronal rather than polyneuritic.

W. K. HUNTER.

**THE PATHOLOGY OF SO-CALLED ACUTE MYELITIS.** By  
(16) H. DOUGLAS SINGER, *Brain*, Summer 1902, p. 332.

IN this paper the author records two cases of what he considers typical examples of acute myelitis. The first, a man aged 56, had contracted syphilis two years before the onset of his symptoms.

The symptoms set in somewhat suddenly with numbness and tingling in the legs, followed in a few hours with complete flaccid paraplegia, anæsthesia (as high as eighth dorsal root), and retention of urine. In a few days the flaccidity gave place to a spastic condition of the legs, and there was now incontinence of urine and fæces. Later, bed sores and cystitis supervened, and the patient died eight weeks from the onset of his illness.

On examining the cord a lesion was found involving the eighth, ninth and tenth dorsal segments. The Marchi stain showed practically all the nerve elements at this level to have undergone degeneration. Above and below the lesion there was ascending and descending degeneration.

The blood-vessels at the seat of the lesion showed a typical syphilitic arteritis. Both internal and external coats were greatly thickened, and in places the lumen of the vessel was quite obliterated. In the larger vessels there was a small-celled infiltration into the perivascular spaces, and there was increase of nuclei both in the internal and external coats. There was no increase of the neuroglia elements to be seen in the sections, and no infiltration of inflammatory cells. The condition was therefore a chronic inflammation of vessels, with subsequent obliteration of their lumens, and necrosis or degeneration of the nerve tissues which these vessels supplied.

The second case, a boy, aged 12, was the subject of congenital syphilis. The paraplegia here also developed rapidly in the course of two or three days, and death occurred about seven weeks later. In this case the lesion was in the sixth dorsal segment. Microscopically the vessels were found to be precisely similar to those in the first case, though the lumen of none of the larger vessels was quite obliterated. There was some overgrowth of the perivascular connective tissue, but no appearance of inflammatory cells in the spinal cord itself. The vessels above and below the lesion were practically normal.

The author also refers to nineteen other cases of myelitis, in fifteen of which there was a definite history of syphilis. He concludes as follows:—“(1) So-called acute myelitis is found on microscopic examination, in the majority of recorded cases, to be not inflammatory, but due to thrombosis of spinal vessels. (2) That by far the most common cause of this thrombosis is syphilitic arteritis; and that senile arterial degeneration forms a considerable proportion of the remaining cases. (3) This view as to the pathology is confirmed clinically by the analogy between this disease and cerebral thrombosis.”

W. K. HUNTER.

**PATHOGÉNIE DE CERTAINES CAVITÉS MEDULLAIRES.**

(17) Par A. THOMAS and G. HAUSER, *Rev. Neurolog.*, Oct. 30, 1902, p. 957.

THE case on which the observations are based was that of a woman æt. 73, who presented weakness of the right arm and leg, but who did not show any of the sensory phenomena characteristic of syringomyelia. There was no muscular atrophy, and the knee-jerks were diminished. Examination of the spinal cord exhibited a bilateral lesion ; on the right side from the level of the lower end of the medulla to the conus terminalis, and on the left side as far down as D ix. In the upper cervical region the lesion was a cavity formation occupying the fore-part of the posterior columns and commissure. Lower down the lesion was limited to the posterior horns. In the upper dorsal region, the posterior, lateral and anterior horns were replaced by neuroglial tissue. But on the left side numerous oblique or transverse fissures divided and altered the posterior horn. In the upper cervical region the lesion histologically resembled that of syringomyelia, but in the other parts of the cord were to be found irregular foci and artefacts with degeneration of the nervous elements and arterio-sclerosis, foci of sclerosis surrounded by a zone of neuroglia, and fissures, occupying usually the axis of the posterior horns. The blood-vessels chiefly affected were the artery of the posterior median septum and of the postero-lateral fissure.

The conclusions at which the authors arrive are, that the fissures and foci of degeneration are essentially of vascular origin, and are due to arterial obliteration or from the pressure of perivascular œdema. Similar instances of this have been already described by various authors, more especially by M. Dejerine in cases of syphilitic myelitis. At the periphery of the softened areas, the neuroglia reacts, as around an irritative lesion, and gives rise to sclerosed zones which mask the original loss of tissue. The case therefore shows that vascular disturbances may be the starting-point of softening in the spinal cord, and that areas of softening so produced may give rise to a neuroglial overgrowth.

W. A. TURNER.

- ON A CASE OF CONGENITAL PORENCEPHALUS, IN WHICH**  
 (18) **THE PORENCEPHALIC AREA CORRESPONDED TO THE**  
**AREA OF DISTRIBUTION OF THE LEFT MIDDLE CEREBRAL ARTERY.** By D. A. SHIRRES. *Studies from the Roy. Victoria Hospital, Montreal*, vol. i. No. 2, Jan. 1902.

THIS account contains a history of the case in which it is shown that the patient, a female, died at the age of 76 from pneumonia, and had been, during a great part of her life, apparently a normal individual, except that the right limbs were slightly weaker than the left.

The condition is thought to have been due to an embolism of the left middle cerebral artery at the eighth month of development.

A large porencephalic cavity was found in the left hemisphere, replacing the third frontal convolution almost entirely, the posterior third of the middle frontal, the lower two-thirds of the pre-central and post-central convolutions, the supra-marginal and the angular, the first temporal and the posterior half of the second temporal.

There is an elaborate description of the atrophies and degenerations found in other parts of the central nervous system, which included atrophy of the right half of the cerebellum, of the left optic thalamus, the left corpora quadrigemina, red nucleus, and left optic nerve.

In the cord the white matter showed absence of the right crossed and of the left direct pyramidal tracts. In the grey matter some of the motor cells were apparently absent, and there was "an abnormal arrangement of the nuclei of the back muscles."

The writer found in the mid-brain, pons and medulla, evidence of old degenerative changes in many tracts, associated with the lesion of the cortex.

The details are too numerous for description here, and are, on the whole, in accordance with our present knowledge of the connections of the tracts involved. The paper also contains a discussion on primary and secondary degeneration, on the fillet, on tracts in the ventro-lateral region of the cord, on the ætiology of spasticity, with a brief resumé of the literature of these subjects.

DAVID WATERSTON.

- GLI ASPERGILLI NELL' ETIOLOGIA E NELLA PATOGENESI**  
 (19) **DELLA PELLAGRA.** C. CENI, *Riv. Speriment. di Freniatria*, 1902, f. 2-3.

It has long been recognised that pellagra is in some way dependent upon the consumption of immature and badly preserved maize, but nevertheless the essential pathology of this very fatal,

though generally slowly progressive, disease has hitherto remained obscure. It was known that aspergilli were present in the bad maize, but there seemed to be no ground for attributing to these organisms any pathogenic importance in pellagra, for they could not be found in any numbers in the tissues, and, moreover, they were regarded as non-pathogenic, with the exception of *aspergillus fumigatus*, which, however, was recognised to be the cause of an entirely different malady, namely a form of pseudo-tuberculosis affecting especially the lungs. Nevertheless, Ceni, by a series of magnificent experimental investigations, has succeeded in demonstrating that aspergillar infection is the essential cause of pellagra. He has even pushed his researches into the domain of therapeutics, and obtained satisfactory evidence that the disease is amenable to treatment by serum of goats and horses immunised to the infective agents. The importance of these discoveries will perhaps be better understood when it is mentioned that in Italy alone about 60,000 persons at present suffer from pellagra.

The author summarises the conclusions that may be drawn from his observations as follows:—(1) Persons who die with the characteristic phenomena of pellagra, of the acute or sub-acute form, almost always succumb in consequence of an aspergillar infection. This infection, generally localised to the lungs, pleura, pericardium and pia mater, constitutes the determining cause of the said morbid phenomena. (2) The pathogenic agents of this infection are especially *aspergillus fumigatus* and *aspergillus flavescens*, which usually act alone, very rarely being present together to form a mixed infection. (3) These parasites pass from the intestine in the form of spores, become localised in various organs and tissues that suit them, and there elaborate and emanate very virulent toxins which give rise to phenomena of general poisoning, as well as to local inflammatory processes of a diffuse character. (4) The gravity of the morbid phenomena of this pellagrous aspergillosis has commonly direct relationship to the pathogenic power of the two above-named species of aspergilli, and to the state of their virulence. The times at which this aspergillosis affects the human subject with the gravest and most characteristic symptoms correspond to the annual life-cycles of the two aspergilli. (5) Maize food-stuffs infected by aspergilli constitute a direct joint cause of this aspergillosis, as they serve as a vehicle for the passage of the said parasites from the environment to the human organism by way of the intestinal tract. (6) Aspergillar infection of maize and maize food-stuffs is intimately related to bad sanitary conditions of the places in which these substances are prepared and kept.

This brief summary of conclusions cannot convey an adequate idea of the width of the basis of original observation upon which



they are founded. The author's systematically arranged and clearly stated account of his work occupies exactly 100 pages. Perhaps one of the most praiseworthy portions of his investigation is that which served to reveal the fact that these aspergilli are greatly increased in virulence when grown upon maize. He attributes this increase in virulence to the circumstance that the conditions of growth are such as to favour the formation of spores. He finds that the acute forms of pellagra are generally due to infection by *aspergillus fumigatus*, the sub-acute and chronic forms to infection by *aspergillus flavescens*. The more severe recrudescence of the disease which takes place in spring corresponds to the period of greatest natural development of *aspergillus fumigatus*, the less severe recrudescence in autumn is coincident with the period of greatest natural development of *aspergillus flavescens*. Ceni's investigations show that whilst the common aspergillosis (pseudo-tuberculosis) is determined by invasion of the tissues by the fully developed organism which is comparatively innocuous, pellagrous aspergillosis is dependent upon the presence of the parasite in the spore form, in which it elaborates very virulent toxins. His experiments with *penicillium glaucum*, which has been alleged to be the essential pathogenic agent in pellagra, gave negative results. His observations upon experimental immunisation and serum therapeutics are to be fully dealt with in a future paper.

W. FORD ROBERTSON.

**L' IPOFISI NEL MIXEDEMA E NELL' ACROMEGALIA.** G.  
(20) VASSALE, *Riv. Speriment. di Freniatria*, 1902, f. 2-3, p. 25.

In this paper the author, firstly, discusses the question of the significance of the enlargement of the pituitary body that is commonly, though not constantly, found post-mortem in cases of myxœdema and of acromegaly; and, secondly, defends his own previously stated conclusions regarding the functional importance of this organ against certain recent criticisms. He thinks that, on the ground of the observations that have been made, it must be regarded as determined that the increase in the size of the pituitary body in myxœdema and in acromegaly is a purely functional hypertrophy dependent upon the circumstance that a greater call has been made upon it to perform its specific function in consequence of some unknown changes of metabolism associated with these diseases. In myxœdema the enlargement is not compensatory for abolition of the thyroid function. The characteristic change in the organ in acromegaly is not an adenomatous one, as has been asserted. He cites a case to show that true adenoma of the hypophysis may occur without acromegaly, and he endorses the con-

clusion of Caselli, based upon a careful examination of the recorded cases, that there is no evidence to prove that other tumours of the organ are associated with this disease.

The author's experimental observations upon the effects of extirpation of the hypophysis, carried out some years ago in conjunction with Sacchi, seemed to prove that this organ is essential to life, complete removal producing a definite series of morbid phenomena, terminating in death in a few weeks. Partial destruction of the gland did not cause similar phenomena. Confirmatory evidence was obtained by Gatta, Kreidl and Biedl, and Caselli. More recently, Friedmann and Maass, and Lo Monaco and van Rymberck, have carried out some experiments from the results of which they conclude that animals are able to tolerate complete extirpation of the hypophysis, and that the organ is not therefore one of vital importance. Vassale criticises the methods and conclusions of these observers. He especially points out that in the few successful results alleged to have been obtained, means were not taken to ascertain definitely that all pituitary tissue had been removed or destroyed.

W. FORD ROBERTSON.

### CLINICAL NEUROLOGY.

#### UEBER DIE VERSCHIEDENHEIT DER PROGNOSE DER

(21) **PLEXUS UND NERVENSTAMMLÄHMUNG DER OBEREN EXTREMITÄT.** Von L. BRUNS, *Neurol. Centralbl.*, Nov. 16, 1902, S. 1042.

THE author has analysed his cases of single peripheral nerve palsy and plexus paralysis from the point of view of prognosis. From a total of 133 cases he has selected 70 which meet the requirements necessary for statistical purposes. Of these, 47 were peripheral nerve paralysees, all but 8 (7 peroneal and 1 sciatic) involving individual nerves of the upper extremity. In the remaining 23 cases the paralysis was dependent on a lesion of the brachial plexus. In all the cases analysed the paralysis was due to trauma, using the word trauma in its widest application. Cases where a nerve was completely divided are not included. All were treated with electricity for a considerable period. The exact etiology of the 70 cases is given in tabular form.

Of the 47 cases of peripheral nerve paralysis, 31 (66 per cent.) recovered; while of the 23 brachial plexus palsies, recovery only took place in 6 (26 per cent.). The large percentage of recoveries in musculo-spiral paralysis, 19 of 22 cases (87 per cent.), is striking.

In only one of 4 serratus magnus cases did the paralysis disappear. Of 7 cases of obstetrical plexus paralysis, in not one did recovery take place. The author points out that in his experience the prognosis in a case of traumatic peripheral nerve palsy is two and a half times as favourable as it is in the case of a plexus paralysis.

The explanation of this difference is difficult to find. B. deals with this question at considerable length, and suggests that the cord itself or the nerve roots in close relation to it may be damaged more frequently than has hitherto been supposed in these so-called brachial plexus paralyses. It still remains to be definitely shown why brachial plexus lesions should occupy as regards prognosis a position midway between peripheral nerve lesions, where the outlook is generally favourable, and lesions of the spinal cord, where the reverse is the case.

EDWIN BRAMWELL.

**A STUDY OF LANDRY'S PARALYSIS; WITH A REPORT ON  
(22) THREE NON-FATAL CASES.** By THEODORE DILLER, *Journ.  
Nerv. and Ment. Dis.*, Oct. 1902, p. 577.

THE earlier pages of this paper are devoted to a rough review of the prominent clinical features recorded in the literature of Landry's Paralysis with the object of showing that most observers have drawn their own conclusions, often at variance with those of others, as to what symptoms may and what symptoms may not be legitimately included in a description of this disease.

The author then briefly records three cases of ill-defined disease which he assumes on somewhat slender grounds to be examples of this condition. The third of these cases is remarkable for the apparently important part played by syphilis as a causal agent, and the influence of this and other infective processes in the production of Landry's paralysis is made the subject of some discussion.

After reference to the recorded anatomical findings in cases which have come to autopsy, Dr Diller expresses the opinion that the name Landry's Paralysis should not be restricted to the symptom-complex described by Landry in 1859, but should be held to embrace all examples of acute motor paralysis which stand midway between Anterior Poliomyelitis and Multiple Neuritis, and which frequently present strong resemblances to one or the other of these morbid entities.

E. FARQUHAR BUZZARD.

**SCLÉROSE EN PLAQUES INFANTILE A FORME HEMIPLEGIQUE**(23) **D'ORIGINE HÉRÉDO-SYPHILITIQUE PROBABLE.** ParGEORGES CARRIER, *Rev. Neurolog.*, Oct. 15, 1902, p. 929.

THE case recorded by the author is that of a child who was probably tainted by hereditary syphilis, but who reached the age of seven years without displaying any signs of disease beyond some mental inaptitude. She then developed a sudden but transitory right brachial monoplegia, which was followed three years later by a slowly progressive right hemiplegia and aphasia. Her intellectual weakness became more marked, and epileptic seizures, commencing in the right leg, began to occur at intervals of about a month. With the exception of some intention tremor and exaggerated reflexes on the left side of the body, all the symptoms pointed to a local lesion in the left rolandic area, and the skull was opened over that region by M. Picqui. Nothing was found but a slight thickening of the pia over the operculum. This was removed, but the child died a few hours later.

A careful macroscopical and histological examination of the central nervous system revealed numerous patches of sclerosis scattered through the white matter of the brain and spinal cord. The meninges were not thickened, but the vessels displayed everywhere considerable increase in size of the inner and middle coats. The pyramidal cells of the cortex were diminished in size and number and showed signs of atrophy with the Nissl stain.

The author agrees with Moncorvo in thinking that hereditary syphilis plays an important part in the production of disseminated sclerosis in young children.

E. FARQUHAR BUZZARD.

**UEBER DIE UNTER DEM BILDE DER MYELITIS TRANS-**(24) **VERSA VERLAUFENDE MULTIPLE SKLEROSE.** VonE. FLATAU und J. KOELICHEN, *Ztschr. f. Nervenheilk.*, Bd. xxii. Heft 4, 1902, p. 250.

AFTER referring briefly to cases by Pitres, Siemerling and Nonne, in which disseminated sclerosis had strongly simulated transverse myelitis as far as clinical features were concerned, the authors come at once to the real subject of their paper.

A woman, sixty years of age, who had previously enjoyed very good health, after some exposure to cold became in the course of a few days almost completely paralysed in the lower extremities. The onset was associated with pain and fever, and, although no disturbance of sensation supervened, the loss of power and of reflexes in the legs, together with incontinence of the sphincters

and the absence of any symptoms higher up, all pointed to a diagnosis of acute myelitis in the lumbo-sacral region of the cord. General wasting of the lower limbs and the development of bed-sores preceded the death of the patient, which took place three and a quarter months after the onset of her illness.

At the autopsy no macroscopical changes were observed in brain or cord; the histological examination appears to have been confined to the latter part of the nervous system. Nissl, Marchi, Weigert, carmine and hæmatoxylin methods of staining were used, and the authors give a very careful and detailed description of the pathological changes. They found throughout the whole length of the cord scattered foci of sclerosis in various stages of development, and drew their deductions as to the pathology of the condition from a minute study of the different microscopical appearances in the recent as compared to the more advanced patches of disease.

The authors unhesitatingly come to the conclusion that the morbid process is essentially an inflammatory one, and that it has its origin in the blood-vessels—in other words, there is some morbid agent present in the blood which sets up inflammation, and the first evidence of this inflammation is seen in the degeneration of the myelin sheaths of the neighbouring nerve fibres. They agree with most modern observers in regarding disseminated sclerosis as a form of diffuse myelitis, characterised by great resistance to the disease on the part of the nerve cells and their axis cylinders, and consequently by the absence of any marked secondary degeneration. A short digest of other published cases of acute or subacute insular sclerosis is given in the latter part of the paper.

E. FARQUHAR BUZZARD.

**A CASE OF FRIEDREICH'S ATAXIA.** By R. S. C. EDLESTON,  
(25) *Brit. Med. Jour.*, Nov. 22, 1902, p. 1642.

THE patient, a female, was under observation for five years. Since her seventh year awkwardness in walking and indistinctness of speech were noticed, and later some tremor of the arms on movement. At fourteen, after an attack of measles, her condition became aggravated. On examination the cranial nerves were found normal; the pupils reacted to light, and neither nystagmus nor retinal change were present. Voluntary movements were accompanied by inco-ordination and tremor. Gait was reeling. Romberg's sign was present. Knee and ankle jerks were absent; skin reflexes and sensation were normal. There was definite lateral curvature of the vertebral column and talipes equino varus

Five years later these symptoms were further advanced, and the patient almost helpless. Her mental condition remained good. Nystagmus was still absent. T. GRAINGER STEWART.

**HEREDITARY CEREBELLAR ATAXY IN TWO BROTHERS.** By (26) J. MITCHELL CLARKE, *Brit. Med. Journ.*, Nov. 22, 1902, p. 1640.

THE patients were two brothers, aged twenty-three and twenty-six. No heredity was traceable. The previous health of each was good, and neither showed evidence of syphilis. In both the condition followed injury. The younger brother two years previously fell 20 feet, was apparently uninjured, but next day tremor of arms and trunk, increasing on movement appeared. A year later there was stiffness of the right leg, and giddiness, followed six months later by numbness. The elder brother when twenty-five hurt his right knee, and shortly after affection of walking, stiffness of the right leg, and unsteadiness manifested themselves. In both cases the initial symptoms were similar. Increased knee and ankle jerks, nystagmus, optic atrophy, reeling gait, and, in the younger brother, affection of speech and tremor were present. The exceptional features were lateral scoliosis, pes cavus, and the presence of Babinski's sign on the more affected leg, physical signs which rather belong to Friedreich's disease.

T. GRAINGER STEWART.

**A BRIEF REPORT OF THE CLINICAL, PHYSIOLOGICAL, AND (27) CHEMICAL STUDY OF THREE CASES OF FAMILY PERIODIC PARALYSIS.** By JOHN K. MITCHELL, SIMON FLEXNER, and D. L. EDSALL, *Brain*, Spring 1902, p. 85.

THE authors describe two new cases, mother and daughter, and also give some further details of a case published by Mitchell in 1899. The attacks in the new cases were quite typical in character, but are of interest in that (1) the mother's father and his brother had been subject to similar attacks; (2) the mother's attacks were increased by too much or too rich food and by eating sweets; (3) in neither were the attacks brought on by exercise even to fatigue; (4) the attacks began at an early age in both, viz., four and six years respectively.

The experimental results were mainly negative. The alkalinity of the blood was practically normal. Blood serum injected into the general circulation, the femoral artery, the sciatic nerve, the lumbar spinal canal, and the cranial subdural space of rabbits had

no effect upon the knee-jerks and yielded no evidence of increased toxicity.

The urine was not hypertoxic, but possessed a high degree of acidity which was not due to acetone, diacetic acid, nor lactic acid. The ammonia in one case was a little less than normal. Kreatinin, estimated by the Neubauer-Salkowski method, was much diminished for two or three days before an attack, rising in amount either during or subsequent to the attack with a sudden bound to about normal, and falling again as suddenly two or three days before the next attack. From this the authors argue some defect in metabolism which they suggest may have its seat primarily in the muscles themselves.

The stomach contents examined during an attack on one occasion showed complete anacidity and cessation of all digestion.

H. DOUGLAS SINGER.

**ZUR KENNTNISS DER RÜCKENMARKS-TUMOREN.** Von  
(28) MEYER, *Ztschr. f. Nervenheilk.*, Bd. 22, H. 3 und 4, S. 232.

THE author describes the clinical condition of a girl who suffered from a tumour pressing upon the lower part of the cervical region of the spinal cord. No member of her family had shown signs of syphilis or tuberculosis, nor had the patient suffered from any disease of importance. She had twice fallen on to her left shoulder, the second time only a year before the onset of the symptoms. At the age of fourteen she began to suffer from severe pain in the left shoulder, quickly spreading to the left arm, and always worse at night. A sensation of "deadness" in the arm quickly came on, followed by weakness and atrophy of some of the muscles, especially of the extensors of the fingers. Fifteen months later there came on spastic weakness of the left leg, followed by a similar affection of the right leg. Spontaneous pain and tenderness of the spine in the middle cervical region and numbness and weakness of the opposite arm appeared; whilst the paraplegia progressed until no voluntary movement remained, although painful flexor spasms frequently occurred. The anæsthesias were difficult to map out, but the ulnar fingers, the ulnar sides of both forearms and arms were early affected, whilst some affection of sensibility over both legs came on later in the disease. Other symptoms were headache, vomiting, and occasional double vision; no optic neuritis was present. There was no spinal deformity. The patient died two and a half years after the onset of the symptoms, and at the autopsy there was found a smooth, hard fibro-sarcoma, almost the size and shape of a hazel-nut, and attached to the fifth and sixth left cervical posterior root ganglia.

The tumour had grown partly within and partly outside the theca, and several adjacent nerve roots were flattened out in their intrathecal course. The left half of the spinal cord was severely pressed upon and showed marked degenerative changes, but the right half was but little affected. The author discusses the differential diagnosis of the case, both as to the nature and the seat of the lesion. On account of the difficulty of excluding pachymeningitis hypertrophica, and the weak condition of the patient when first seen, no operation was suggested. A dilated stomach found at the autopsy is suggested as the cause of the vomiting; no mention is made of the presence or absence of optic neuritis at a late stage, nor as to the post-mortem condition of the optic discs; nor does the author suggest any cause for the double vision from which the patient occasionally suffered.

STANLEY BARNES.

**BEITRAG ZUR DIAGNOSTIK DER GESCHWÜLSTE DES**  
 (29) **STIRNHIRNS.** S. AUERBACH, *Ztschr. f. Nervenheilk.*, Band 22, 1902, p. 312.

AN unmarried lady, forty-eight years of age, who from youth had suffered from recurrent attacks of migraine, developed a remarkable change in character, becoming irritable, obstinate, and strangely lazy in her household duties. After a temporary improvement the psychical symptoms, especially the apparent laziness, became more marked than ever. The headaches became more frequent, vomiting was superadded, and she took to her bed and refused to leave it, occasionally passing urine and dejecta under her. The apathy and somnolence persistently increased. No convulsions occurred, nor was any sensory or motor paralysis to be detected. The optic discs were healthy and the superficial and tendon reflexes of the limbs were normal. Her facies strongly resembled that of myxœdema, and this idea was apparently supported by the mental symptoms, and by the existence of six months' amenorrhœa and feelings of chilliness. Accordingly treatment by administration of thyroid gland was undertaken for a time and with some apparent improvement. Suddenly, however, three weeks before death, the patient had an apoplectiform attack, in which she was unconscious, pale and collapsed. Immediately after this, double optic neuritis appeared, more marked on the left side, also left exophthalmos, and almost complete blindness in both eyes. The pupils became practically insensitive to light and convergence. The spinal muscles were also observed to be weak. Headache and vomiting were by no means urgent, and the patient remained singularly indifferent to her illness in spite of the blindness.



After a second attack of collapse the neck muscles were stiff and the head retracted. There was no aphasia nor weakness of any limb or of the face. At the close, pulmonary consolidation, fever and coma developed. The total duration of the symptoms was seventeen months.

At the autopsy, the frontal gyri were somewhat flattened, the dura mater was abnormally adherent to the basis cranii anteriorly, and a firm, encapsuled, spheroidal tumour, the size of an apple, divided by a furrow into two lateral halves, was found situated mesially a short distance in front of the corpus callosum, and implicating the basal two-thirds or so of both frontal lobes, extending on each side as far out as the anterior perforated spots, and backwards close to the optic chiasma. The flattened olfactory bulbs were spread out on the under surface of the growth. (Smell was tested a few days before death and found normal.) Microscopically, the tumour was a fibro sarcoma, probably starting from the dura mater. The ventricles of the brain were not dilated.

PURVES STEWART.

**PARADOXICAL PSEUDOHYPERTROPHY FOLLOWING IN-**  
**(30) FANTILE CEREBRAL HEMIPLEGIA.** By L. PIERCE CLARK,  
*Jour. of Nerv. and Ment. Dis.*, Nov. 1902, p. 641.

IN introducing an account of some cases of infantile cerebral paralysis, in which a slight degree of hypertrophy was present in certain muscles on the paralysed side, reference is made to the fact that no special article on this subject exists in literature although instances of its occurrence have been mentioned by Bernhardt, Gowers, Oulmont, Goudard, Schieber, Kaiser, and the author of the paper under review. Of the 12 cases recorded by the different observers referred to, athetosis was present in 8—in 1 the leg only partook in the athetosis and hypertrophy—in the other 4, attacks of Jacksonian epilepsy occurred. In none of the cases was the family history in any way significant, and the hemiplegic attack occurred after a time of normal health. In no case was paralysis present at birth. In 2 cases out of the 12 the paralysis at its onset was unaccompanied by convulsion or serious constitutional disturbance. In 7 cases the right side was affected, in 5 the left. The time that elapsed between the paralysis and the detection of the hypertrophy was variable—the interval extending from eight to twenty-nine years, and the age at which the hypertrophy was recognised ranged from twelve to thirty-six years. The hypertrophy in such cases may involve all the parts, even the breast and testicles may enlarge. The enlargement has been in the upper extremity in every instance, except one case of

Bernhardt's already alluded to, in which the athetosis and hypertrophy were restricted to the lower limb. The arm has been most enlarged in 9 cases, the forearm in 2, the calf in 1. The muscles most frequently hypertrophied are the biceps, deltoid, and triceps, in that order. The circumferential enlargement does not usually exceed half an inch. Histological examination in two of Dr Pierce Clark's cases showed true hypertrophy of muscular fibres and an increase in the number of muscle spindles. Bony hypertrophy has also been found—the bones usually found enlarged being those corresponding to the overlying hypertrophied soft parts. Dr Pierce Clark suggests that the association of the peculiar cortical lesion with some degree of post-hemiplegic disorder of movement may account for the hypertrophy, for it is significant that in 8 of the recorded cases athetosis was present, and in the other 4—all recorded by Dr Clark,—convulsive seizures, confined in 3 to the paralysed and hypertrophied side, in the fourth affecting that side first and chiefly, occurred frequently.

JAMES TAYLOR.

**WEITERES ÜBER DIE ASTHENISCHE LÄHMUNG, ETC.** Von  
(31) S. GOLDFLAM, *Neurol. Centralbl.*, Juni 4, 1902, p. 390.

THE disease more generally known in this country as Myasthenia Gravis, Goldflam prefers to designate Asthenic Paralysis, therein following Fajersztajn and Kalischer. He believes that this malady is more common than is generally thought; more common, in fact, than the genuine bulbar paralysis of Duchenne. Particulars of eight cases are given, with comments thereon. The first case is that of a man æt. 25 (some notes of which the author had already published), who succumbed to the disease. A searching examination of the brain, spinal cord and nerves, by means of the most recent staining methods, failed to discover any morbid change. The patient suffered from lympho-sarcoma of one lung; and two years before his death, excised portions of the left deltoid showed collections of small cells between the muscle-fibres. After death several muscles were examined, and they all showed a similar infiltration. Goldflam regards these changes as metastatic. He refers to a case of myasthenia observed by Laquer, in which post-mortem no morbid changes were found in the nervous system, but in which the thymus gland was the seat of a lympho-sarcoma, and the muscles infiltrated, as in his own case; and to a doubtful case of the same disease (recorded by Weigert) in which there was a similar mediastinal tumour, without, however, any infiltration of the muscles; reference is also made to a case (observed by Senator) of multiple myeloma, in which there was weakness of the tongue, and

difficulty of swallowing; but this, Goldflam does not regard as an instance of genuine myasthenia. Cases of myasthenia in which innocent tumours were present, such as lipoma of the kidney and dermoid cyst of the ovary, have also been recorded, so that, as Oppenheimer had already remarked, new growths complicate myasthenia with peculiar frequency. Goldflam finds it difficult to explain this connection, especially in the case of innocent tumours, which, as far as is known, yield no toxins. The author remarks of this patient that on the whole the Mya. R. was independent of the symptom of fatigue.

The second case—that of a man æt. 27—is remarkable for the rapidity with which the symptoms attained their maximum—*i.e.* within a fortnight he died from dyspnœa and paralysis of respiration. The third case was that of a woman æt. 22. Her father died of *tumor cerebri*. There were apparently complete remissions which lasted for periods varying from months to three and a half years. She married and had children, each pregnancy having a favourable influence on the disease: so much so, that she declared that “to be always well she would require to be always pregnant.” Patient suffered from muscular twitchings, especially of the face, but not from “fibrillar” twitchings. In the fourth case (woman æt. 32) there was facial hemiatrophy and exophthalmic goitre. In this case the disease, though it probably existed previously in a latent form, ran a *foudroyante* course, causing death within a fortnight. Goldflam, in commenting on it, insists that every instance of isolated ptosis that cannot be otherwise explained should make one think of myasthenia. He remarks on the fact that in Graves’ disease, from which this patient suffered, paralysis, especially of the eye-muscles, may occur without any discoverable lesion of the nervous system to account for it. In the fifth case the intensity of the symptoms would vary even during the course of a single examination. There was also the interesting circumstance that fatigue of one set of muscles induced fatigue in another set. Fine fibrillary twitchings and chronic contraction of the facial muscles were observed. The next case was that of a boy æt.  $4\frac{3}{4}$  years, who had previously suffered from convulsions. There was a neuropathic family history. Talking caused a weakness, not only of the speech muscles, but of the levatores palpebrarum, showing, as in the last case, the reciprocal influence of one set of muscles on another—a phenomenon which Goldflam regards as peculiar to myasthenia. The seventh case—that of a man æt. 61—was remarkable from the fact that the symptoms were slight, and limited to the muscles supplied by the bulb. The myasthenic symptoms entirely cleared up, the patient dying some years afterwards from another disease. The last case—a man æt. 35—was exceptional as showing paralysis of accommodation; the left pupil was at times somewhat dilated.

The fact that, in myasthenia, lasting paralysis attacks those muscles which are in constant contraction—*e.g.* the levatores palpebrarum and the muscles which support the head—is referred to; and Murri's observation is confirmed, to the effect that exhaustion through voluntary contraction has no influence on Faradic response, and, contrariwise, muscles all but entirely exhausted by voluntary contraction respond to the will. Regarding the "seat of the disease," Goldflam considers the facts are against its being situated in the peripheral neuro-muscular apparatus; and he does not think that the possibility of a cortical origin can be altogether excluded, arguing that the peculiar grouping of the myasthenia (*i.e.* the paralysis of entire limbs, or section of limbs, *e.g.* the upper arm or the thigh) points rather to a cortical lesion than to a lesion of lower motor-centres. Regarding the toxic origin of the disease, he failed to find evidences of abnormal toxicity of the urine in the one case he examined for it. The prognosis must be regarded as very grave, most of the cases succumbing later or sooner to the disease.

HARRY CAMPBELL.

**THE CLINICAL HISTORY AND SYMPTOMS OF ONE HUNDRED  
(32) AND TWENTY CASES OF EXOPHTHALMIC GOITRE.\***

By GEORGE R. MURRAY, *Lancet*, Dec. 13, 1902, p. 1612.

Of 120 personally observed cases of exophthalmic goitre, 10 were men and 110 women, a proportion of 1 to 11. The age of onset varied from 15 to 65, but the greatest liability to the disease existed in earlier life from 15 to 35. The disease had not occurred in either parent in any case, but in the brothers and sisters of several patients. An accident or grief, either sudden or prolonged, appeared to be an exciting cause in some cases. Enlargement of the thyroid gland was present at some time during the illness in 117 cases. In 14 a simple goitre had been present before the development of other symptoms for periods varying from 3 to 34 years. In 27 cases the enlargement was slight, in 36 it was moderate, and in 20 it was considerable. The frequency of the pulse varied from 90 to 200. In 66 cases it was between 120 and 150. Great variation in the pulse-rate was sometimes observed in the same case within a short time. Cardiac murmurs occurred rather frequently. In 79 out of 114 cases exophthalmos was observed, in 3 it had previously been present, and in 32 it was absent. Von Graefe's symptom was present in 36 out of 91 cases. A fine tremor was observed in 111 cases, in 4 it was absent, and in 5 it was not noted. In 70 cases a condition of

\* Read in full at a meeting of the Royal Medical and Chirurgical Society on October 28th, 1902.

nervousness was observed, in 3 it was absent, and in the remaining 47 it was not noted. Insanity had occurred in two cases, and in two others hallucinations were described. Sudden attacks with great intensification of the symptoms for several hours occurred in several cases. Weakness of the legs was complained of by 15 patients. Five felt as if the legs would give way, in 7 the legs actually gave way and the patients fell. The skin was damp in 76 cases, and more or less pigmented in 22. There was loss of hair in 10 cases, liability to diarrhoea in 35, and of 19 cases in which the urine was examined, albumen was found in 4 and sugar in 3. Irregularity of menstruation occurred in 23 of 110 cases. Loss of weight was noted in 45 cases. Out of 40 cases 7 died, 2 remained stationary, and 31 progressed favourably while under observation; of the latter 9 practically recovered, 8 were greatly and 14 slightly improved. For treatment in severe cases rest in bed for several weeks was recommended, and if there was much wasting, "Weir Mitchell" treatment. For less severe cases a quiet life, spent chiefly in the open air. Gentle faradism to the neck for an hour night and morning was found to be beneficial. The most useful drugs were belladonna, bromides, convallaria, arsenic, thymus, and supra-renal tablets.

AUTHOR'S ABSTRACT.

**ZUR PSYCHOLOGIE DER MOTORISCHEN APRAXIE.** Von A.  
(33) PICK, *Neurol. Centralbl.*, vol. 21, Nov. 1, 1902, p. 994.

THIS is a report on the case of a man, sixty-two years of age, who for the preceding three years had had, at irregular intervals, curious attacks, of which the patient himself gave the following description. Quite suddenly he found himself unable to speak the words he wished, or do as he desired. Instead of this his words were all topsy-turvy, he could not read nor write, everything swam before him. There was no paralysis and no loss of hearing. In three days usually he was well again. Similar attacks, but with varying individual symptoms, recurred about once a fortnight, and were associated with slight tonic spasms in the region of the masseters. On one occasion he saw a mist or fiery rings. As they continued to persist he became afraid that such attacks were premonitory of insanity, and consulted a specialist. One of the latest is described in detail. A discussion of the psychical phenomena then follows, in accordance with which the case is regarded as one of motor apraxia. Atheromatous degeneration of the vessels with senile brain atrophy is the probable cause of the symptoms.

JAS. MIDDLEMASS.

**UEBER MYOTONISCHE PUPILLENBEWEGUNG.** By ALFRED  
(34) SAENGER, *Neurolog. Centralbl.*, Sept. 16, 1902, p. 837.

SAENGER confirms Strasburger's observation of sluggishness of dilatation and contraction of the pupil, and mentions that he first observed it in November 1901, and had intended to describe the phenomenon in the next volume of *Die Neurologie des Auges*, but that the article on the pupil is postponed to vol. v. His case was that of a married woman, æt. 34; no history of syphilis. Her father died paralysed, and she had one sister insane. Married twelve years ago, and ten years ago she noticed the left pupil became dilated. Recently suffered from diffuse headache, sometimes followed by vomiting. Neither pupil reacted to light, the left being larger than the right. On convergence and accommodation the left pupil slowly contracted, remaining so for a few minutes, then slowly dilating again. With screwing up of the eyes tightly the left pupil similarly contracted slowly, but not so much or for so long. The right pupil was about one-half the size, also fixed to light, but reacted quicker to accommodation and convergence than the left. This one also remained contracted for some time and then dilated quite slowly.

No loss of power of accommodation. Tendon reflexes all normal, the knee-jerks especially being brisk. Normal sensation, except for slight ulnar analgesia. She complained occasionally of shooting "rheumatic" pains, and in view of the Argyll-Robertson pupil present, Saenger considered it to be probably a slowly-developing case of tabes.

Very interesting was the fact that the patient could tell by her own sensations whether the left pupil was dilated or contracted, feeling a painful sensation on the left side of the head when it was contracted.

When the pupils were examined by means of the Westien corneal-microscope, the left was found absolutely fixed to light, with the exception of slight hippus movements in parts of the circumference; the right showed by this means distinct slight movement to light.

Saenger suggests that the anatomical site of the lesion in this phenomenon is not central, but peripheral in the iris, and compares the condition to the myotonic muscular condition in Thomsen's disease. Piltz, he says, has already shown myotonic pupil reaction to occur in the reflex iridoplegia of general paralysis, and also in these cases on tightly closing the eyes, that is, the orbicularis reaction. Saenger thus shows the same thing to occur with accommodation and convergence.

WILFRED HARRIS.

**DIE PARADOXE PUPILLENREACTION UND EIGENE BEO-  
(35) BACHTUNG VON VERENGERUNG DER PUPILLEN BEI  
BESCHATTUNG DER AUGEN.** Von J. PILTZ, *Neurolog.*  
*Centralbl.*, 1902, Ns. 20, 21, and 22, pp. 939, 1012, 1054.

IN a series of three articles the author criticises closely a number of cases of recorded instances of paradoxical dilatation of the pupil on exposure to light. He insists that this result is contrary to natural law, and that true instances of it are exceedingly rare. Several cases he considers unproved for various detailed reasons, notably one of Oestreicher's, which he gives no sufficient reason for refusing to accept. In the same way he refuses to accept as genuine the two cases of tabes of Marina and d'Abundo, in which in each case neither pupil contracted to light or accommodation, while one pupil slowly dilated when exposed to light or on closing the eye. He suggests that warming of the conjunctiva by the light, stimulating the sympathetic, accounted for the dilatation in one case, while the "orbicularis reaction," or the pupil contraction occurring on forcible closure of the eyes, might account for the other case, though he does not quote d'Abundo as saying that the lids were tightly closed. He very properly insists on the importance of distinguishing the orbicularis reaction first described by Gifford, that is, the pupil contraction occurring on tightly closing the eyes, with the subsequent dilatation on opening them, from the paradoxical dilatation of the pupil on exposure to light. Another cause of error, he points out, is the associated dilatation of the pupil which occurs on divergence of the eyes, which is apt to occur when a light is approached to the eyes in a case with weakness of the internal recti, a condition usually present in the recorded cases. The reaction of the pupil to accommodation, he shows, may also stimulate the paradoxical reaction, as also may hippus. He quotes two interesting cases of "perverted pupil reaction" of Vysin's, in which the pupils contracted on focussing a distant object, dilating again on accommodating for a near object. He remarks that both these cases were functional disorders, one being a case of traumatic neurosis, the other a case of migraine, in which the phenomenon appeared only during the attacks, which he takes to be evidence of an accommodation pupillary centre in the cortex. He accepts only five cases recorded as genuine cases of true paradoxical pupil dilatation under light, a case of post-syphilitic optic atrophy fully detailed by himself in the third part of his paper, a case of tubercular meningitis by Leitz, Morselli's case of general paralysis, Sillex's case of severe neurosis following a blow on the head, and Bechterew's case of cerebral syphilis. The most frequent source of error is the so-called orbicularis reaction on tight closing of the eyes, while mistakes may also arise through the convergence,

divergence, and accommodation-reactions, and also from hippus, the true paradoxical dilatation under light being exceedingly rare, and only met with in serious organic nervous disease. However, this phenomenon will have to be restudied in the light of Dr H. K. Anderson's observations on the pupil after experimental division of the cervical sympathetic.

WILFRED HARRIS.

**PUPILLENTRÄGHEIT BEI ACCOMMODATION UND CON  
(36) VERGENZ.** Von JULIUS STRASBURGER, *Neurolog. Centralbl.*,  
1902, Aug. 16, und Nov. 16, pp. 738 und 1052.

IN the first paper a case is described of a young man, æt. 17, with probably early disseminated sclerosis, in whom unequal pupils had been noticed since nine years old. The father had been melancholic for some years. Three years ago the boy's right leg became weak. Right knee-jerk increased, with Babinski reflex on right side. Nystagmus toward the left side, and later in both directions. Intention tremor in right hand. No history of syphilis. The right pupil reacted normally to right, both directly and consensually, while the left was completely fixed for both forms of illumination; a condition of unocular Argyll-Robertson pupil. The reactions of the left pupil to convergence and accommodation were fully performed, but slowly, especially the succeeding dilatation. Contraction of the left pupil took twice as long as on the sound side; and dilatation, which was somewhat oscillatory, took ten to twenty seconds, or five to ten times as long as on the right side. After frequently repeated acts of accommodation the reaction became quicker. Moreover, the changes in the shape of the lens seemed to be less quickly performed than normal. Dr Strasburger remarks that as a rule sluggishness of movement and slight alterations in size of pupils are usually found together, but most striking in this case was the marked slowness of movement compared with the considerable changes in size. He remarks that pupil changes are well known to occur in disseminated sclerosis, and mentions a case of König's, who described a somewhat similar change in the pupil of an idiot, probably a syphilitic child, with fixed pupils and slowness of reaction to convergence. He mentions also two cases of reflex iridoplegia in children with slow dilatation of the pupils. In his second paper Dr Strasburger criticises Dr Saenger's recent paper, "Ueber myotonische Pupillenbewegung" (*q.v.*). He objects to the term "myotonic pupil reaction" used by Dr Saenger on the ground that no similarity with the muscle condition found in Thomsen's disease has been proved, and prefers to leave the description of the condition as "sluggishness of the pupil on accommodation and convergence."

WILFRED HARRIS.



**BEITRAG ZUR LOCALIZATION DES CEREBRALEN HEMI-**

(37) **ANÄSTHESIE.** Von K. SCHAFFER, *Neurol. Centralbl.*, B. 21, Nov. 1, 1902, S. 1004.

THE following case is reported. A labourer, eighteen years of age, was taken suddenly ill with shivering, severe headache, and vomiting. The following day he became unconscious, and was taken to hospital. The next day he was conscious, but confused and somewhat incoherent. He complained chiefly of headache, and held his head stiffly backwards. There was hyperæsthesia of the skin, especially of the extremities. The temperature varied between 38·9° and 39·5° C. The diagnosis then made was epidemic cerebrospinal meningitis. On the eleventh day of his illness he woke in the morning, after a quiet sleep, with severe pain in the left leg. Examination discovered complete motor and sensory paralysis of the left half of the body. His mind was quite clear. The temperature gradually fell, and the stiffness of the neck also disappeared. Six months later the patient was transferred to another ward and his condition was then one of complete analgesia and anæsthesia of the left side. There was also no sense of the position of the limbs on that side. About seven months later he died from the effects of aortic incompetence. The brain was very carefully examined and found to present the following condition. A localised softening was present, and was visible on the brain surface, occupying the lowest part of the right anterior and posterior central convolutions, and also the anterior end of the superior temporal. After hardening, the brain was cut in frontal sections, and the softening was found to involve the upper part of the anterior limb of the internal capsule, the body of the nucleus caudatus, the putamen, the insula, and the convolutions above-mentioned. It was carefully noted that the optic thalamus was uninjured by the softening process. There were evidences of secondary degeneration in the thalamus, however, and the lateral ventricle was dilated. There was also secondary degeneration in the pyramidal tract. In the thalamus the degeneration affected the cortico-thalamic neurons. If the area of softening were projected on the horizontal plane it would be found to affect the anterior limb, the knee, and the anterior half of the posterior limb of the internal capsule. The interest of this lies in the clinical fact that besides hemiplegia there was complete hemianæsthesia and analgesia. Another deduction is that an extra-thalamic lesion can cause permanent hemianæsthesia.

JAS. MIDDLEMASS.

**PSYCHIATRY.****ZUR PSYCHOPATHOLOGIE DER NEURASTHENIE. Von A.**(38) PICK, *Arch. f. Psychiat.*, Bd. 35, H. 2, 1902.

PROFESSOR PICK here relates the symptoms of a case of what Morel called "délire émotif." The patient was an old woman of seventy-nine, of good intelligence, and showing only the physical symptoms of senility. There was no evidence of hysteria. All her life she suffered more or less from mental pain, often over the most trivial affairs. If her husband stayed from home later than usual she got into such a state of apprehension that she used to go to the police to ask them to search for him. As she reached old age this state became so pronounced that she had to be placed under care. In such cases it is noticeable that only painful impressions are morbidly exaggerated. The author regards them as an exhibition of neurasthenia.

JAS. MIDDLEMASS.

**EIN FALL VON CEREBROPATHIA PSYCHICA TOXÆMICA**(39) (KORSAKOFF), **GASTRO-INTESTINALEN URSPRUNGES.**EMIL RAIMANN, *Monatsschr. f. Psychiat. u. Neurolog.*, Oct. 1902.

THIS case related to a man who had suffered from indigestion, diarrhoea, and precarious appetite, for two years. In January 1902 he became worse and showed, besides the above-mentioned symptoms, giddiness, inability to walk, nystagmus, and disturbances of speech of bullar origin. At the same time he became somnolent and apathetic, with confused ideas of time and place, and marked loss of memory.

These mental symptoms increased as the case progressed.

Post-mortem examination showed much congestion of the pia-arachnoid membrane, and the grey matter of the brain. Around the optic motor centres there was much congestion, with numerous round cells, and some blood exudation.

The intestines were bound together, and there was some purulent effusion between them; the mucous membrane of the small intestine was thickened. Peyer's patches were enlarged and pigmented, and there were numerous ulcers with infiltrated, but not undermined, edges. On the mucous membrane of the jejunum there were many small growths, some of them reaching the size of a hazel-nut, with ulcers in their centre in many cases.

The large intestine was thickened and pigmented; there were numerous small hæmorrhages, with small patches of necrosed mucous membrane over them.

The Mesenteric glands were enlarged, and there was central necrosis in some of them.

The author described this as a case of lympho-sarcoma of the follicles of the mucous membrane of the small intestine, with secondary growths in the mesenteric glands.

He suggested that there was a causal relation between the intestinal lesion and the psychic condition, that this psychic condition was, in fact, due to an auto-intoxication by poison absorbed from the intestinal tract.

He quoted another case of a girl, in whom constriction of the pylorus and dilatation of the stomach resulted from the swallowing of sulphuric acid, and this physical condition was followed by a stuporose mental state, which he attributed to an auto-intoxication from the absorption of poison derived from the septic contents of the dilated stomach.

R. G. Rows.

**IMPRESSIONI DI UNA RAPIDA VISITA AD ALCUNI MANI-  
(40) COMII DELLA SCOZIA. L. BIANCHI, *Ann. di Neurolog.*,  
anno xx. 1902, f. 4.**

IN the autumn of last year, Professor Bianchi of Naples visited several representative Scottish asylums, and also one of the largest of those in England. He has now recorded, in the journal of which he is the editor, the impressions he has formed of our asylum system. The article is obviously written solely for the information of his compatriots, but it contains observations and criticisms which, coming from so high an authority, may well be pondered by all those in this country who are responsible for the proper care and treatment of the insane, or who are honestly endeavouring to grapple with the grave and difficult problem of how to lighten the still increasing burden of lunacy.

Professor Bianchi commences by telling his countrymen that in his opinion the Scottish asylums he has visited are, in respect of their sumptuousness and the amount of liberty accorded to the patients, greatly superior to any others in Europe. At the same time, it is easy to discern in the enumeration of details exemplifying the former feature, a feeling that the thing is greatly overdone. The author is, however, content to attribute it to the practical spirit that pervades these institutions, to a certain industrial pomp that is inherent in the British character, and to that genuine, enduring and practical sentiment of charity which is, he says, in no other people so strongly developed.

A short but strictly accurate account is next given of the general characteristics of the asylums he visited, the provision made for the needs, comforts and amusement of the patients, the

arrangements that obtain in regard to attendants, the most prevalent forms of insanity, etc. He remarks especially upon the small proportion of general paralytics and the large number of cases of phthisis in the Scottish as compared with the Italian asylums. Up to this point the tenor of his comments upon what he saw is one of hearty commendation. Passing next to the sanitary service, he says that in general it is not organised with corresponding breadth of view. The medical superintendents are too much occupied with administrative duties; the number of assistant medical officers is far too small, the average number of patients under the charge of each being from 300 to 400, or even more. He remarks that it is not difficult to understand that under these circumstances, however desirous the doctor may be to devote his energies to the examination and physical and moral treatment of his patients, it is practically impossible that he should be able to get to know so large a number thoroughly and to govern them rationally. An assistant medical officer should not have more than 120 or 130 patients under his care. He indicates that there is at present passing through the Italian parliament a bill which, in one of its provisions, makes a reasonable limitation of this nature. Quoting accurate figures, he gives it as his opinion that there is too great a difference between the salaries of the medical superintendents and those of the assistant medical officers.

The author then deals at considerable length with the present position of research upon the pathology of insanity in this country. The time at his disposal did not permit of a visit to the Claybury laboratory, and it is therefore easy to understand how the general impression he received of the provision made in our asylums for the prosecution of scientific research seems to have been that it is of a primitive nature and inadequate for modern requirements. He speaks with approval of the laboratories seen at two of the asylums, but chiefly on the ground of the guarantee they give of progress in the future. He remarks that he can think of no field of research more fruitful and more promising of useful applications to social hygiene and to the prophylaxis and treatment of mental diseases, than that of the etiology and pathogenesis of insanity. Of the Laboratory of the Scottish Asylums he says much that ought to be gratifying to its promoters. He states, however, that though it is well organised, it is not yet provided with the various means of research with a liberality proportionate to the richness of the asylums. It is evident, he says, that the administrators still show timidity and hesitation in providing those means of scientific research that have the appearance of a luxury, or that seem to them not to be of immediate utility. The microscope, normal and pathological anatomy, bacteriology, chemical pathology, photography and experiments upon animals are not, in his opinion,

scientific luxuries, but means indispensable for the attainment of the ends aimed at by all well-organised and well-managed asylums. To him asylums are not merely houses of refuge for the mentally afflicted, but at the same time centres for investigation into the causes of mental disorders. The fact that most forms of acute insanity depend either upon auto-intoxication, exogenous toxins or infections, justifies the creation of pathological laboratories attached to asylums, for when the etiology and pathology of mental diseases and of psycho-somatic degeneration shall have been more fully elucidated, these disorders will have their rational therapeutics and prophylaxis. He says that nothing should be grudged to these laboratories, neither the best men nor the necessary equipment. Italy with its limited means has already achieved far more in this direction. Its asylums are in great part provided with laboratories. The provincial asylum of Naples has from the beginning had its laboratory, under the direction of Professor Armani. He approves of the idea of a central laboratory for the Scottish asylums, but criticises the existing working arrangements of the institution. He would practically limit the work of its director to that of research. He disapproves of the system of sending tissues from a distance for investigation and report. The pathologist ought himself to make autopsies upon all cases that he is to investigate, and he ought to be afforded facilities for making bacteriological observations upon the living subject. He should, in a word, be placed in more favourable conditions in order that he may be enabled to utilise the available clinical and pathological material to the greatest advantage.

W. FORD ROBERTSON.

## TREATMENT.

**TREATMENT BY THE TOURNIQUET TO COUNTERACT THE**  
**(41) VASOMOTOR SPASM OF RAYNAUD'S DISEASE. H.**  
**CUSHING, *Jour. of Nerv. and Ment. Dis.*, Nov. 1902, p. 657.**

THE removal of a tourniquet from a limb is invariably followed by a flushing of the limb peripheral to the site of constriction—this active hyperæmia being presumably due to temporary paralysis of vasomotor control caused by the constriction. This suggested the employment of the tourniquet as a means of counteracting the local conditions of vasomotor spasm in Raynaud's disease, and the excellent result obtained in one case by the frequent employment of this procedure leads the author to think that it may have a curative effect, either by producing subsidence of the undue irritability of the vasomotor centre—according to Raynaud, the

basis of the spasm—or, more probably, because the local vascular conditions in the extremities become so altered by the periods of active circulation, that slight peripheral stimuli (*e.g.* cold) no longer provoke the intense reflex constrictor responses.

The case referred to, in which this method of treatment was tried with apparently complete success, was that of a woman, aged thirty-five, who had suffered for many months from a marked condition of local asphyxia of all four extremities, with exacerbations of almost daily occurrence requiring the use of morphia; slight superficial patches of gangrene had affected fingers and toes, and, as a result, most of the finger-tips were tapering. The daily application of the flat rubber bandage to one or another limb according as the symptoms indicated, for a period of one or two up to five minutes or longer, gave pronounced relief to the burning pain from the first; the attacks became less and less frequent and severe, her general condition rapidly improved, and she was quite free from all symptoms about five weeks after the commencement of treatment. Several months later, she was threatened with a recurrence of the trouble in her hands, but a few applications of the tourniquet gave immediate and complete relief.

A. W. MACKINTOSH.

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# **Review** **of** **Neurology and Psychiatry**

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## **Original Articles**

### **URBAN SELECTION AND MENTAL HEALTH.**

By JOHN MACPHERSON, M.D., F.R.C.P.E.

THE modern tendency of the population of Western Europe to flock into large cities forms one of the most complex problems with which the Sociologist has to deal. It is regarded popularly with disfavour, and as a sign of the increasing love of excitement and pleasure which can only end in the degeneracy of the race. Fortunately we have evidence which enables us to regard the question from a purely scientific standpoint, and which tends to show that for their better subjection to the influence of the great law of natural selection such a movement of the people is necessary for the development of the race. With considerable suddenness the people of Western Europe have passed from under the sway of feudalism, a more or less continuous condition of internecine war, and the influence of vast epidemics of a deadly nature, which were permitted to exhaust themselves for want of further pabulum, into a condition of comparative freedom, of almost universal peace, and so general an immunity from epidemics that natural selection in the latter respect might almost be considered to have ended but for the prevalence of alcoholism, syphilis, phthisis, and the epidemic diseases of

children. Further, the last invasion of Europe from the East is hundreds of years old, and no fresh infusion of racial elements, owing to the sharp political boundaries, can be expected so long as the "balance of power" remains as at present. Invasion being out of the question, there would be an apparent danger of the stagnation of the more or less crystallised racial elements of Europe, were it not that "migration," a newer and far more powerful solvent of races, has supplanted it. In order that migration should take place at all there must exist more or less powerful inducements, and in order that it should successfully attain its object—the subjection of the peoples to a stringent natural selection—it must be from the rural into the densest centres of town population. On the economic side, the inducements to migrate are the advantages of co-operation, higher wages, steady work and the chances of advancement. On the social side, the excitement of the crowd, individual expansion and superior comfort. The effect of these two sets of combined motives is to produce a steady flow of the rural population into the towns in every civilised European country. The following tabular statement shows what the result of this migration has been upon the population of Scotland for the past forty years :—

	1861.	1871.	1881.	1891.	1901.
Urban,	1,138,189	1,918,670	2,306,852	2,672,715	3,120,241
Rural,	1,924,110	1,441,348	1,428,721	1,394,932	1,351,862

The changes in England are correspondingly striking :—

	1861.	1871.	1881.	1891.
Urban,	12,696,520	14,929,283	17,636,646	20,802,770
Rural,	7,369,704	7,782,983	8,337,793	8,198,248

What applies to Great Britain applies generally though in varying degrees to France, Germany, Austria, Italy and the United States of America. In 1860 the population of Rome

was 184,000, in 1894 it was 450,000. During the same period Vienna and Christiania have trebled their populations. Copenhagen has increased two and one half times, and Stockholm has doubled its population. This growth of city population is almost wholly at the expense of the rural population, for the town birth-rate is not so much in excess of the mortality as to account for more than a fraction of this increase. In view of this extraordinary movement of population the question naturally suggests itself—What is its result upon the mental and physical health of the people? It must, one would suppose, be either destructive, and tend towards degeneration, or through the “narrow gate” of natural selection lead to a marked variation in the type of the race.

That a marked physical change of type occurs as a result of urban selection has been shown by numerous observers. Those interested in this subject will find a succinct sketch of the work in Ripley's “Races of Europe,” p. 537 *et seq.*

First of all there is to be observed the tendency towards dolicocephaly which has been clearly established by Ammon of Carlsruhe among the inhabitants of the State of Baden, and independently by Lapouge of Montpellier among the inhabitants of southern France. Various explanations are forthcoming to explain this phenomenon. Ammon has accounted for it on the supposition that the long headed races—the Teutonic and the Mediterranean—display a greater tendency to migrate into cities, and his observation has been dignified by the name of “Ammon's law.” It is further held by Ammon that the broad headed Alpine race, more conservative and less adventurous, prefers to remain upon the land. This explanation has been rightly disputed, and it is believed by other workers that the struggle for existence in town life results in the natural selection of the dolicocephalic race over the brachycephalic, which latter is obviously unfitted to survive under the new conditions. At any rate it is found without exception that the preponderance of dolicocephalism over brachycephalism holds true in Paris, Lyons, Vienna, Munich, Rome and Madrid. The cities of the British Isles defy comparison, for the preponderating element of dolicocephalism in the country prevents, according to the methods as yet adopted, the discovery of any perceptible change in the shape of the head.

A second physical result of great importance is the variability of stature produced by town life. The observations are in one sense contradictory, but in another they appear to be conclusive. In the cities of Belgium, Baden, Switzerland and Hungary the town populations exceed in height those of the surrounding districts. Later observers in Germany, Spain, France and Great Britain have produced precisely opposite statistics. Thus the inhabitants of Edinburgh and Glasgow are four inches shorter than the surrounding population and thirty-six pounds less in weight. But a most important fact in this connection is that the people of the West or aristocratic parts of almost all European cities distinctly exceed in stature the inhabitants of the East or tenement portions.

The explanation of this fact is to be found only in the operation of the law of natural selection, for even granting that many of these favoured people are the descendants of taller immigrants to the towns, it shows their inherent capability of surviving the depressing influences of town life. But it is inconceivable that the population of Western London, for instance, have all immigrated from the country to the richer part of the metropolis without first, in the persons of their ancestors, having had to undergo the selective process of life in the less favoured portions of that city.

The third physical characteristic which falls to be noticed with regard to town populations is their distinct tendency to brunetteness. Even in Northern Germany, which is peopled by a preponderating blond population—fair-haired and blue-eyed—this brunetteness of the town inhabitants is clearly apparent. This fact of itself is sufficient to overthrow the arguments of the upholders of any ethnic theories and to substantiate the contention that natural selection alone is the agent which operates in the production of the urban type. Pigmentation is undoubtedly an index of vitality and probably conduces to the superiority of the brunette type in the competition of urban life.

The selected urban type then is dolicocephalic, tall, slimly built, and inclined to be dark-haired and dark-eyed. Mentally, it is vigorous, quick-witted, capable of endurance, and self-controlled. Fond of amusement, it is not readily overcome by excitement, and does not yield itself easily to excesses. As regards alcohol it ought to have undergone a special evolution,

for this selection is a particularly stringent one. How different is the case with the immigrant fresh from the country. The sordid process of evolution is before him and his descendants, if he shall have any. Ere they can attain to the level of the preceding type they must run the gauntlet of zymotic disease, alcohol, syphilis and phthisis.

The direct application of the foregoing principles to the purpose of this paper must be approached through the vital statistics of a country. Naturally I turn to Scotland, and for purposes of comparison select two districts, namely, the three north-western counties of Sutherland, Ross (with Cromarty), and Inverness, on the one hand, and the south-western group of Renfrew, Ayr and Lanark on the other. I shall call the former the north-western and the latter the south-western group.

The north-western group is chiefly rural and the south-western chiefly urban, as is shown in the following table taken from the census figures for 1901 :—

	North-Western.	South-Western.
Urban . . .	31,524	1,602,078
Rural . . .	156,470	260,697

Not only so, but while the smaller population of the north-western group has fallen in fifty years by 17,000 persons, the south-western group has increased by upwards of 980,000 persons, an increase which can only be accounted for by immigration. The former is an example of a rural population being drained by migration, and the latter an example of an urban centre being fed from rural populations.

Some of the vital statistics of these two groups are of importance. Thus in 1899 the percentage of births to the total population in the north-west group was 2·25, in the south-western it was 3·33; for the same year the percentages of deaths were respectively 1·71 and 1·96; and of marriages 0·44 and 0·84 respectively. In connection with marriage it is interesting to observe a decreasing disinclination towards it in the northern group, where 52 per cent. of the men and 49 per cent. of the women over fifteen years of age were unmarried in 1899 as against 46 per cent. of each sex in the southern group. But the chief fact which it is desired to bring into prominence as illustrative of the greater stress of natural selection in the urban

district is, that while in 1899 the mortality under five years of age in the north-western group was only 18·5 per cent. of the total deaths, in the south-western group it was 42 per cent.

I now turn to the ultimate question of the production of insanity within the foregoing specified districts.

*Annual Average Number of Pauper Lunatics placed in Asylums per 100,000 of Population.*

	Group I.			Group II.		
	Sutherland.	Ross.	Inverness.	Ayr.	Lanark.	Renfrew.
1880-4	44	42	56	48	60	56
1885-9	47	49	57	50	57	53
1890-4	62	52	71	56	61	68
1895-9	89	60	78	62	62	71

The significance of the above table may be obtained less accurately but more concisely from the following statement :—

*Proportion (per 100,000 of Population) of Pauper Lunatics annually placed in Asylums in the years 1892-1901.*

All Scotland.	North-Western Group.	South-Western Group.
58	Sutherland . . . 77 Ross and Cromarty 61 Inverness . . . 76	Ayr . . . 55 Lanark . . . 57 Renfrew . . . 63
Average 58	Average 71·3	Average 58·3

The numbers sent to asylums are chosen because they form the only reliable index for comparison and because they indicate the acute occurring insanity of the districts. In dealing with the above figures we are faced with the following problem :— Why, notwithstanding the presence in the southern group of agents which we know to be active exciting causes of insanity,



and the comparative absence of these agents in the northern group, is acute insanity which requires asylum care apparently more prevalent in the latter group? Unfortunately the statistics of alcoholism in this country are not available, nor under the present system of collecting such facts can we hope for accurate information. We may, however, compare our southern group with Paris, the population of which city does not so much exceed it, nor are the habits of the peoples with regard to alcohol so divergent as to place comparison out of the question. M. Paul Garnier, Physician to the Special Infirmary for the reception of the insane within the Prefecture of the Seine, reported that the number of alcoholics received had increased by 143 per cent. in fourteen years; and that out of a total number of 2859 admitted into this infirmary in one year, 40 per cent. of the men and 12 per cent. of the women were alcoholics (La folie à Paris, Congrès 1890). Similar figures are forthcoming from all the chief European cities where an effort is made to furnish reliable statistics on such subjects. Without any certainty to the contrary we may not flatter ourselves that our urban populations are more sober than similar aggregations on the continent of Europe. General paralysis is a rapidly increasing affection in the larger Scottish urban communities, but like alcoholism it is to all intents and purposes practically, and so far as regards the present object, a negligible quantity in the remoter rural districts. In the Forty-third Annual Report of the General Board of Lunacy for Scotland, p. lvi., it is shown that the proportion of deaths of pauper patients from general paralysis per 1000 of the average number resident is for asylums serving large towns 22·6; for asylums serving urban and industrial districts 11·2; and for asylums serving rural districts 5·1. Besides general paralysis there are other manifestations of cerebral syphilis or para-syphilis, chiefly arterial, which result in premature decrepitude of the nervous system, and which are particularly observable in the urban asylums. Without going further into detail it may, I think, be taken as granted that the conditions of urban life create a distinct body of insanity which does not exist in the quieter rural communities. Why then is not the order reversed, and why does not the proportion of occurring insanity stand higher in the urban districts? In order to remove some of the difficulties and misconceptions which surround the problem, it

will be necessary to glance again at the rural district under review. In every population which is being rapidly depopulated, just as in a pond or river in which the water level is steadily falling, a stage is reached at which previously concealed inequalities and disagreeable objects reveal themselves. If instead of calculating the existing insanity upon the present emaciated population, which is only its brother, so to speak, we calculate it a generation or two generations farther back upon a fuller population, which was its parent and grandparent, we should, I think, be estimating more justly, though not perhaps more legitimately. On the other hand a population may be so sparse as to prevent the effect of sexual and natural selection from operating. Such, it is understood, has been the case in some portions of the Western States of America, where it is believed by some to account for the very high proportion of insanity and nervous disease reported to exist in these districts. But so long as a rural community is increasing by excess of births over deaths, or even maintains its numbers, or has its emigration counterbalanced by immigration, it does not as a rule manifest any undue disproportion of insanity to population compared with other similar districts.

If insanity were, as many people suppose, a malady for the occurrence of which individuals were to a large extent morally responsible, it should be as rare in placid rural communities, removed from the temptation of destroying vices, as swallows in winter. But as insanity is merely a sign or a symptom of the action of slow natural selection, and as rural communities are not exposed to the more rapid agencies of the same law, which can only operate effectively in densely populated centres, it follows that insanity should be expected to be more prevalent in a community where natural selection for any reason is weak. For the same reason an urban population should exhibit less of what is ordinarily designated insanity. In the first place its population is selected, for it is being constantly recruited by immigrants who must after all possess at least two qualities, namely, the enterprise to migrate and an absence of conspicuous eccentricity; it is also selected in another sense by the exigencies of town life, for a town population is the survival of a huge infant mortality. Finally it must, gradually, by the elimination of the unfit, and by the

survival of the more fit, attain to a position in which the slow natural selection of insanity shall cease to be necessary. No doubt in the process, which so far as we can see has no prospect of ending, much mental and nervous disease is produced and made conspicuous, such as alcoholism, general paralysis, syphilis of the nerve centres, etc., etc. If we only knew a little more of the pathology of the ordinary forms of acute insanity it would undoubtedly become apparent to us that they also, like the three diseases just mentioned, are but chips in the laboratory of nature wherein is being shaped the destiny of the human race.

### **A STUDY OF THE MODES OF ONSET IN EIGHTY CASES OF DISSEMINATED SCLEROSIS.**

By ASHLEY W. MACKINTOSH, M.A., M.D.,  
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It is perhaps more true of disseminated sclerosis than of any other chronic nervous disease that the diagnosis is difficult in the earliest stages: for this reason, a study of the modes of onset noted in a large series of cases may be not only interesting but of considerable practical value. The difficulty of early diagnosis is due mainly to two facts, first, that the modes of onset are so many, so varied and, often, so similar to the symptom-complex of other recognised diseases; and, second, that the disease may run its course for months or years before any of the so-called "cardinal" symptoms appear—if ever they do appear. A study of such a series of cases, as that whose analysis is given below, makes it certain that, ere long, it will be possible and even necessary to widen our views regarding the "cardinal" symptoms of disseminated sclerosis, along such lines as those followed by Buzzard, specially, among English writers.

The variability of the modes of onset of disseminated sclerosis has been recognised since the days of Charcot's Lectures. The initial symptoms may be spinal (most often) or cerebral or bulbar (seldom). Again, the onset is usually gradual, but it may be acute or even sudden. The legs are usually affected before the arms, but the opposite may occur. Motor or sensory symptoms may lead; ataxia or tremor—generally a later symptom—may,

however, be the first sign. Literature contains not a few instances of all these and many other varieties in the modes of onset of this disease.

A few years ago I had the rare opportunity of tabulating the records of Dr Ferrier's \* extensive series of cases of disseminated sclerosis: these, together with a few cases seen more recently, form the basis of the following statistics and remarks regarding the modes of onset and the earliest signs of this disease. In the absence of autopsies, I have felt it necessary to include only such cases as exhibited well-known cardinal symptoms: this has necessitated the exclusion of many of the most interesting cases, which are almost certainly true examples of the disease, belonging to the ever-widening family of "formes frustes" or anomalous types, with which the writings of Charcot and others have made us familiar.

Subjected to the test mentioned, the series is reduced to 80 cases: in 67 of these (*i.e.* 83·75 per cent.) tremor of the arms was present, while ataxia or slow movement of the arms was noted in other 5 (6·25 per cent.); nystagmus occurred in 65 (81·25 per cent.), optic atrophy in 38 (47·5 per cent.), and scanning speech in 16 (20 per cent.).

#### *Modes of Onset in Eighty Cases.*

[*Note.*—In the following classification, it may be stated that, as will be seen, the groups are not mutually exclusive: in many cases, a combination of symptoms was present at the onset, and these cases will therefore appear in two or more groups. The same remark applies to the later list of the earliest signs noted.]

##### *A. Gradual motor onset—21 cases.*

###### *a. Beginning in the legs.*

(1) In 4 cases the first symptom was weakness of one leg, followed by weakness of the other leg at intervals of 1 to 3 years, tremor of the arms appearing in 1½ to 4 years after the onset. One case had also incontinence of urine at the onset, and in 2 cases there was transitory dimness of vision.

\* I have pleasure in expressing my indebtedness to Dr David Ferrier for placing at my disposal the great bulk of the material on which this paper is based.

(2) In 10 cases the onset was with gradual weakness and stiffness of both legs—like simple spastic paraplegia.

One case had also vertigo at the onset, another had sciatica-like pains, diplopia and retention of urine, while a third had a tendency to incontinence of urine as an early sign. In many cases no symptom, other than paraplegia, was noted for years, and in many cases also the paraplegia was transitory—disappearing entirely and reappearing later, perhaps after the lapse of years. Thus, in two cases no other symptom was noted till 6 years later, when diplopia (transitory) and gastric crises respectively appeared.

In most of the cases of this group the gait is noted as spastic mainly. Those cases in which an unsteady, staggering gait was one of the most prominent initial symptoms are grouped together later (*vid.* group C).

(3) In 2 cases the first sign was weakness of one leg, followed in the course of some months by tremor of the arm on the same side. In both cases the onset of weakness in a leg was accompanied by a staggering gait.

*b.* Beginning in an arm.

In one case the first sign was transitory weakness of an arm, followed three years later by sudden weakness of both arms, and soon thereafter the legs began to get weak.

*c.* Beginning in one arm and leg at the same time—hemiparesis.

This occurred in 4 cases. In one the face also was affected on the paretic side, in two it was intact, and in the fourth the facial condition is not noted. In one of these cases a gradual right hemiparesis at the onset was accompanied by left internal strabismus—like a pons-lesion; both these symptoms quite disappeared and the right hemiparesis reappeared three years later and again improved. In another case left hemiparesis of gradual onset appeared transitorily, and “shortly after” squint and diplopia (transitory) were noted, while, a year later, right hemiparesis was present and gradually increased.

*B.* Gradual onset with both motor and sensory symptoms—15 cases.

*a.* Beginning in the legs.

(1) In 5 cases the onset was with gradual weakness of one

leg, accompanied by pain (3 cases), numbness (1 case), coldness and tingling (1 case) of the same leg, and followed by similar symptoms in the other leg at varying intervals, *e.g.* 1,  $1\frac{1}{2}$ , 7 years. Symptoms of paresis or tremor of the arms did not appear till later, in one case  $11\frac{1}{2}$  years after the onset. In one of the cases with pain at the onset, this was so severe that tabes was diagnosed, but the classical symptoms of disseminated sclerosis appeared later.

(2) In 3 cases the onset was with gradual weakness of both legs, accompanied by numbness (2 cases), pain (1 case) in the legs. In one of these cases diarrhoea was also present at the onset, and attacks of gastric crises occurred 4 years later; in another the gait was noted as markedly staggering at the onset, and amblyopia occurred about the same time. The arms were affected soon after the onset in one case, but not till 4 and 10 years later respectively in the other two cases.

(3) In one case weakness and numbness of one leg was the first sign, accompanied by difficulty in passing water, and followed in a few months by weakness, "deadness" and tremor of the arm on the same side. [In this case, however, attacks of vertigo probably preceded the onset of these symptoms for 3 months, and there was a history of fits, evidently hysterical, for 3 years before this.]

*b.* Beginning in the arms.

In 6 cases the onset was with weakness and paræsthesiæ of one arm, more especially of the hand, (5 cases), both arms (1 case).

In the case with bilateral onset, gradual paraplegia appeared one year later. In 3 of the cases with unilateral onset, similar symptoms appeared in the leg of the same side at intervals of some months (*i.e.* hemiparetic form); in another, the affection of one arm was followed by a similar affection of the other arm a month later, amblyopia 3 months later still, while gradual paraplegia appeared in about a year. In the remaining case the arm condition was quite transitory, and there was no other symptom till gastric crises occurred 2 years later, followed soon by sudden paraplegia which showed a remarkable number of remissions.

*C.* Onset with ataxia or tremor of limbs—20 cases.

*a.* Beginning in the legs.

In 7 cases the first symptom was ataxia of the legs or "loss of control" over them, and, more especially, unsteady or reeling gait.

In 3 cases unsteady gait was noted before the onset of any other symptom (*e.g.* weakness of the legs). In another case ataxia of one leg was accompanied by a tendency to incontinence of urine, and was followed  $1\frac{1}{2}$  years later by ataxia of the other leg. The other 3 cases have already been referred to—the unsteady gait accompanied gradual paraplegia and numbness of the legs in 1 case, weakness of one leg in 2 cases.

*b.* Beginning in the arms.

(1) In 8 cases the onset was with tremor of one or both hands. In three of these cases—in two of which the tremor was accompanied by paresis of the arms—gradual paraplegia, with staggering gait, was noted later after an interval of 4 months,  $1\frac{1}{2}$  years,  $5\frac{1}{2}$  years respectively. In one case sudden paraplegia appeared  $2\frac{1}{2}$  years after the tremor began. In another case the tremor of the hands was accompanied by vertigo, amblyopia and occipital headache, and the legs became paraplegic  $2\frac{1}{2}$  years later. In the remaining three cases tremor of one arm was followed by affection of the leg on the same side—in one case simple tremor of the leg, and, in the other two cases, tremor and weakness of the leg, with amblyopia on the affected side (discs normal). [In a fourth case, in which the early condition was also one of purely unilateral tremor beginning in the arm, there was a history of numbness of the fingers of the affected side for some months before the onset of tremor—this case is therefore not included here, but is found in group *D*.]

(2) In 5 cases the first symptom noted was ataxia—rather than tremor—of one or both hands, the patient readily "dropping things," etc.

In one case, amblyopia and attacks of vertigo also occurred at the onset. In four cases paraplegia gradually appeared within a year of the onset; in the fifth case the ataxic condition of an arm was accompanied by weakness and tingling of the same part, and followed by weakness of the leg on the same side in six months (included in group *B b*).

*D.* Onset with sensory symptoms—10 cases.

*a.* Onset with paræsthesiæ—7 cases.

In 3 cases attacks of numbness or tingling of the hands occurred for 1, 5, and 9 years respectively before the onset of other symptoms (paraplegia). In one case attacks of loss of sensation and numbness of the legs were the only symptom till paraplegia gradually developed a year later. In another case numbness of the fingers of one hand existed for several months before the onset of tremor of the same part, and tremor of the leg on the same side soon appeared. In another well-marked case attacks of numbness in various parts of the body were a prominent symptom for 15 years before the onset of paraplegia—in this case, however, there was a history of transient amblyopia 3 years before the onset of the numbness. In the remaining case transient attacks of numbness of one half of the body occurred for 11 years before the onset of weakness of the legs, and the arms were not affected till 4 years later still.

*b.* Onset with pains in the legs—3 cases.

In one case crampy, "sciatica-like" pains were present at the onset and suddenly disappeared, to be followed by gradual paraplegia; in another case "lumbago" and a cord-sensation at the waist were prominent before the onset of sudden loss of control of the legs; in the third case attacks of "rheumatic" pains in the legs and the left hand were the first symptoms noted.

*E.* Acute or sudden onset—10 cases.

*a.* Onset with sudden paralysis of one leg—2 cases.

In one case the paralysis was quite transient, and gradual paraplegia followed in the course of a few months. In the other case the leg weakness was permanent, diplopia was noted 2 years later, the other leg got gradually weaker, intention-tremor and nystagmus were not noted till 6 years after the onset.

*b.* Onset with sudden paraplegia—4 cases.

In two cases the paraplegia was accompanied by numbness and loss of feeling of the legs. In one case there was complete recovery in a few hours, and, later, gradual paraplegia developed; in two cases the improvement was much slower and less complete, and the arms became affected in 2 and 2½ years respectively; in the fourth case the paraplegia was permanent

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and accompanied by pain round the waist, speech became indistinct and slow in 3 years, and intention-tremor and nystagmus were noted 7 years after the onset.

*c.* Onset with sudden hemiparesis—one case.

In this case the hemiparesis—face not affected—had quite disappeared in two or three days: there were no other signs till 7 years later, when sudden weakness, unsteadiness and numbness of the legs developed. [In another case, almost certainly an atypical case of disseminated sclerosis, the same mode of onset, with sudden, temporary hemiparesis was noted.]

*d.* Onset with an apoplectiform attack—3 cases.

In one case the attack was slight and was accompanied by only very little paralysis; tremor of the hands followed very soon. In another case unconsciousness lasted for two weeks; weakness of the legs, tremor of the hands and difficulty of speech developed almost immediately afterwards. In the third case the patient, as the result of a severe fright, is said to have lost consciousness or, at least, memory “for months,” and paraplegia and marked intention-tremor of the arms developed as a direct sequela.

*F.* Onset with cerebral symptoms.

Apart from the cases which started with a hemiparesis or an apoplectiform attack (*vid. supra*), certain cerebral symptoms occurred at the onset, either alone or accompanied by symptoms in the limbs, in 19 cases.

*a.* Diplopia at the onset—4 cases.

In one case diplopia occurred along with gradual paraplegia at the onset (included in group *A a*). In another case diplopia and transitory amblyopia of one eye appeared a month before symptoms in the legs. In the other two cases diplopia was the only sign for 1 and 1½ years respectively—in the latter of these cases gradual paraplegia then developed, while, in the other case, the arms were affected two years before the onset of paraplegia.

*b.* Squint at the onset—2 cases.

In both cases an external rectus was weak. In one case left internal strabismus accompanied gradual right hemiparesis at the onset—both symptoms were quite transitory (included in group *A c*). In the other case the onset was very acute—

squint, amblyopia (progressive later) and "dancing of letters on reading" (nystagmus found) were noted as the initial signs.

*c.* Nystagmus at the onset—2 cases.

This is usually a late sign, but in the case just quoted it was one of the initial signs. In a second case it was the only sign noted—most marked after dancing—for 4 years, when the legs became affected.

*d.* Vertigo at the onset—4 cases.

In all cases vertigo accompanied initial signs in the limbs, except perhaps in the case included in group *B a* (*q.v.*), in which attacks of vertigo occurred two or three times a day as probably the only sign for 3 months.

*e.* Headache (occipital) at the onset—1 case.

Along with vertigo and amblyopia, occipital headache accompanied tremor of the hands at the onset (included in group *C b*).

*f.* Amblyopia at the onset—12 cases.

In 6 cases amblyopia accompanied symptoms in the limbs at the onset; in another case diplopia also was present; and, in another, squint and nystagmus—these 8 cases have already been referred to. In the remaining 4 cases amblyopia was noted for a period of three months up to six years before the appearance of other symptoms. One of these cases exhibited, as the only sign for six years, progressive amblyopia, first of the left eye and then of the right eye, with marked optic atrophy; then the right arm and leg began to get weak and numb and, at the same time, movement of the eyes to the left became defective (pons-type), while tremor of the right arm appeared later.

It is perhaps worthy of note that—although often late in their course—10 of these 12 cases (*i.e.* 83 per cent.) showed definite signs of optic atrophy, while the condition of the discs is not mentioned in the other two; in the whole series of 80 cases, optic atrophy, as already stated, was found in 38 cases (47·5 per cent.).

In the following summary of the earliest symptoms in the series of 80 cases, it will be understood that, in the vast majority of cases, these are the first symptoms *as noted by the patient*; many symptoms, *e.g.* objective sensory defect, nystagmus, optic atrophy, etc., may have existed unobserved by the patient before

the onset of such obvious symptoms as paresis, numbness, pains, diplopia, etc.

*The Earliest Symptoms Noted in the Series of Eighty Cases.*

[*Note.*—As stated in a former note, the groups are not mutually exclusive.]

I. Motor Paresis in 45 cases.

Paresis of both legs (paraplegia) .	17 (4 sudden)
„ one leg . . . . .	14 (2 sudden)
„ one arm . . . . .	8
„ both arms . . . . .	1
Hemiparesis . . . . .	5 (1 sudden)

II. Sensory Symptoms in 29 cases.

Paræsthesiæ of hands . . . . .	10
„ „ legs (especially feet) .	10
Pains in legs . . . . .	7
„ hand . . . . .	1
Girdle-sensation . . . . .	3
Numbness of trunk . . . . .	2

III. Ataxia or Tremor in 20 cases.

Ataxia of legs or unsteady gait .	7
Ataxia or tremor of arms . . . .	13

IV. Sphincter Troubles in 5 cases.

Incontinence of urine . . . . .	3
Retention of urine . . . . .	2

V. Apoplectiform attack in 3 cases.

VI. Cerebral Symptoms (other than hemiparesis or apoplectiform attack) in 19 cases.

Amblyopia . . . . .	12
Vertigo . . . . .	4
Diplopia . . . . .	4
Squint . . . . .	2
Nystagmus . . . . .	2
Headache (occipital) . . . . .	1

*Remarks.*—One may remark specially on the following points brought out by the foregoing analysis :—

- (1) The extraordinary variability of the modes of onset.
- (2) The comparatively large number of cases with *acute* or *sudden* onset—10 cases out of 80, *i.e.* 12·5 per cent. Besides

these 10 cases, there were at least 4 cases in which sudden paraplegia (3 cases) or sudden loss of control of the legs (1 case) was the first sign in the legs, although not the first symptom of the disease. Another case had transitory paresis of one arm, and the next sign was sudden weakness of both arms 3 years later. Some cases, although not actually sudden in onset, progressed very rapidly: thus in one case, within 3 months of the onset, all the cardinal symptoms were very pronounced, and the gait was so reeling that the patient could not stand.

(3) The frequent occurrence of purely *unilateral* symptoms at the onset:—

Weakness of leg, followed by tremor of arm on same side, 2 cases.

Weakness	{	of leg,	"	{	tremor	of arm	"	"	1 case.
Numbness									

Weakness	{	of arm,	"	{	weakness	of leg	"	"	3 cases
Numbness									

Tremor of arm,	"	tremor of leg	"	"	2 cases.
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Tremor of arm,	"	{	tremor	{	of leg	"	"	2 cases.
			weakness					

Gradual hemiparesis (face affected in one)	.	.	4 cases.
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Sudden hemiparesis (face not affected)	.	.	1 case.
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Thus, 15 cases out of 80 (18·75 per cent.) exhibited unilateral symptoms at the onset.

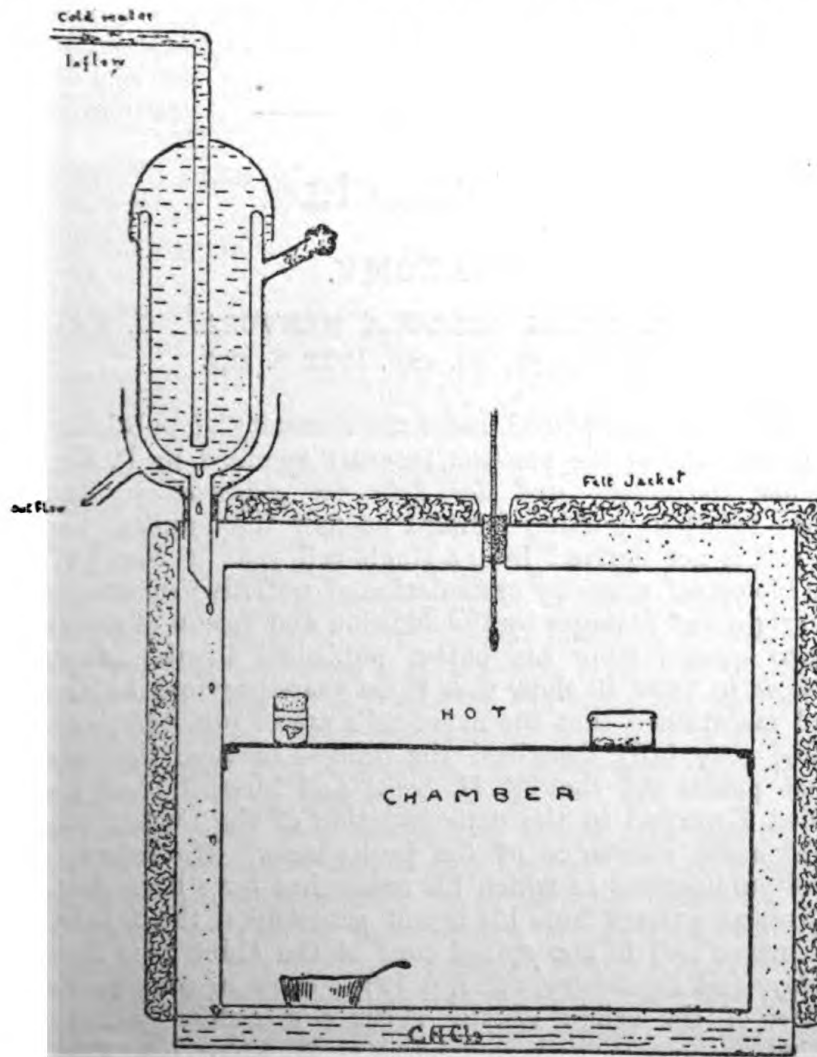
(4) The comparative frequency of the occurrence of *sensory* symptoms at the onset, either alone (10 cases), or combined with other symptoms (19 cases). In all these cases the sensory disturbances were purely subjective. Objective defect of sensation was found early in the course of not a few cases, but it is manifestly impossible to say—except, perhaps, in very rare instances—that this was present at the very onset when such symptoms as numbness, pains, etc., would naturally become obtrusive. The old teaching was that the *absence* of sensory symptoms was characteristic of disseminated sclerosis: the writings of Erb, Oppenheim, Freund, etc., have disproved the truth of this. Certainly, my series of cases shows clearly that the occurrence of sensory disturbances, either in the history of a case or in the record of the present condition, is one of the most common and most valuable auxiliary symptoms, as suggesting a diagnosis of disseminated sclerosis in cases which start under the guise of

some other disease, in which sensory disturbance plays no part (*e.g.* spastic paraplegia).

### AN IMBEDDING OVEN WITHOUT GAS.

By J. A. MURRAY, M.B.,  
Lanark District Asylum, Hartwood.

MANY workers must have experienced difficulty in obtaining a constant temperature for imbedding ovens, especially where gas is not available or of varying pressure as in many towns. The following arrangement, which I owe to the ingenuity of my brother, Mr Walter Murray, is practical, cheap, and very efficient.



A shallow layer of chloroform is introduced into the jacket surrounding the hot chamber and the only exit is provided with a vertical condenser. Heat is applied below by an oil lamp or other convenient source, and the hot chamber is soon surrounded with chloroform vapour which, as it passes upwards, is condensed and falls back into the jacket, so that the process goes on continuously.

It is advisable to add a small quantity of absolute alcohol to prevent the formation of phosgene from the chloroform, and this has the further advantage of slightly reducing the boiling point ( $61^{\circ}\text{C}$ ). In this way the imbedding oven in use here has, for the last three months, given a constant temperature of  $59^{\circ}\text{C}$ . for weeks at a stretch, and what is very important, has never risen above  $60^{\circ}\text{C}$ .

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## Abstracts

### ANATOMY.

**PER LA GENESI DELLA CELLULA NERVOSA.** O. FRAGNITO,  
(42) *Anatomischer Anzeiger*, Bd. xxii. 1902, S. 292.

THE author has been placed under the necessity of publishing this note on account of the position recently assumed by P. Kronthal (*Von der Nervenzelle und der Zelle im Allgemeinen*, Jena, G. Fischer, 1902) in putting forward as new the opinion that the nerve cell is not derived from a single cell, that it does not grow in the biological sense by assimilation of nutritive substances, but takes origin and enlarges by the addition and fusion of small cells. Fragnito quotes from his paper, published in the *Annali di Neurologia* in 1899, to show that three years previous to Kronthal he had maintained that the nerve cells are of pluricellular origin, and had very fully described the process of their development. He also points out that F. H. Scott and himself have likewise preceded Kronthal in the demonstration of the nuclear origin of the chromatic substance of the protoplasm. He refers to the various publications in which his researches have been described, and gives an extract from his recent paper upon the development of the nerve cell in the spinal cord of the chick (see *Review of Neurology and Psychiatry*, vol. i. p. 19) to show in what particulars his view of the origin of the nerve cell differs from that of Kronthal.

W. FORD ROBERTSON.

**UEBER EINEN BESONDEREN KERN DER FORMATIO RETICU-**

(43) **LARIS IN DER OBEREN BRÜCKENREGION** Von v. BECHTEREW, *Neurolog. Centralbl.*, Sept. 16, 1902, S. 835.

IN this short paper attention is drawn to the presence in man of a nucleus, which the author had already described in the dog and cat, in the last edition of his "Leitungsbahnen, etc." It lies in the tegmentum of the pons close to the middle line, immediately behind the corpora quadrigemina posteriora. The cells which are small compared to their size in lower mammals, where they may be larger than motor elements, lie among the fibres of the continuation of the ground-bundle of the lateral column of the spinal cord, and probably stand in close relation to it. No other connection is described.

The name nucleus centralis superior lateralis, or simply nucleus centralis superior, is proposed for it, and if the latter name be adopted the cell mass which has hitherto borne it may be styled nucleus medianus.

GORDON HOLMES.

**CONTRIBUTO ALLO STUDIO DELLE DEGENERAZIONI CONSECU-**

(44) **TIVE AL TAGLIO DELLE RADICI DORSALI.** TARULLI e PANICHI, *Riv. di Patol. nerve. e ment.*, fasc. 11, 1902.

THE authors cut the posterior nerve roots in cervical, dorsal and lumbar regions of several dogs and examined both central and ganglionic segments by Weigert's and Marchi's methods. In some instances the roots were teased, but in the majority of cases simply examined by section.

As a result of careful systematic examination the writers found that in the posterior cervical and dorsal roots there were a very few degenerated fibres in the ganglionic stump. In the lumbar region the number of degenerated fibres was greater, but that the majority remained unaltered. They therefore conclude that some fibres, especially in the lumbar region, have their trophic centre in the cord, while the large majority take their origin from cells in the root ganglia, as shown by the extensive degeneration in the central stump. It is important to note that in the central stump the number of normal fibres coincides with the number of degenerated ones in the ganglionic stump.

The authors bring their histological results into line with the experimental work of Wersilow and Bayliss, who on stimulating the last lumbar and first sacral roots obtained dilatation of the blood-vessels and rise of temperature in the posterior limbs; while on stimulating the last cervical and the dorsal roots a similar result was obtained but less in degree. T. & P. therefore hold that there exists a relationship between the number of

efferent fibres in the sensory roots and the vaso-dilator effects obtained by their stimulation. They are doubtful if these fibres comprise all the vaso-dilators and think that they are probably joined by others which have their origin in the posterior root ganglia. Supporting this view that some vaso-dilators take origin in the cord, is the fact that after having cut the lumbar roots and some days later having taken away a large portion of the lumbar cord, the vessel dilatation after stimulation is less than that observed in an animal in which the roots only have been cut.

In the cord itself there was also degeneration. In the segment corresponding to the cut root, the writers found in the white substance marked degeneration in the roots at their point of entrance. Here there were very few normal fibres. In sections of the fourth and fifth lumbar the ventral zone of the posterior columns showed a considerable amount of degeneration. Immediately below this region very few degenerated fibres were to be seen in the corresponding zone. In the remainder of the posterior columns there was degeneration, and also in the lateral and in the anterior regions. In this latter the degeneration was very slight. In the grey substance the posterior part was traversed by degenerated fibres, while in the anterior roots some fibres were seen degenerated in the intra-spinal, but not in the extra-spinal part.

On the opposite side of the cord in the white substance a little degeneration was found in the lateral and in the anterior columns.

In the grey matter a few fibres were seen degenerated in the posterior horn and in the intra-spinal anterior root fibres to a very limited extent. In the anterior white commissure there were degenerated fibres running from the posterior horn of the side operated on, to the anterior columns of the normal side.

The authors have found that above the fifth lumbar segment in the ventral zone of the posterior columns there are as many degenerated root fibres as normal ones, but that below that region the fibres are almost entirely intact. They conclude that these latter are of endogenous origin.

Regarding the nature of the degenerated fibres in the intra-spinal portion of the anterior roots, the writers hold the opinion that they are sensory, and that the negative result obtained on stimulating the central end of a cut anterior root is due to the fact that these fibres are very few in number and are also easily exhausted by the operation of cutting.

DAVID ORR.



**NOTE ON THE ARTERIAL SUPPLY OF THE BRAIN IN AN-**  
**(45) THROPOID APES.** By A. S. F. GRÜNBAUM, M.D., F.R.C.P.,  
 and C. S. SHERRINGTON, M.D., F.R.S., *Brain*, Autumn, 1902,  
 Part xcix. p. 270.

THE writers describe a feature of human character not hitherto described in the anthropoid apes, namely, the existence in the cerebral arterial supply of a circulus Willisii resembling that of man. In these apes an anterior communicating artery quite frequently completes the circulus in front.

The observations were made on six chimpanzees and one orang-outang, and in all the specimens but one an anterior communicating artery was found, and in the remaining specimen the anterior cerebral arteries were united by a lateral fusion of the two trunks in the middle line for about four mills.

The observation acquires additional interest from the fact that in no mammalian forms below the anthropoids has this arrangement been found.

The writers tested the freedom of the cross anastomosis of the supply of the cerebral hemispheres by occluding the common carotid artery of one or of both sides, and observing whether this resulted in spasm or in alteration of the excitability to Faradism of the motor region of the cortex; and found that in none of the animals did occlusion of one common carotid produce either effect, while occlusion of both common carotids frequently brought about rapid paralysis of the cortex in the motor region, but in some of the animals no bad effect was detected from an occlusion for eight minutes.

DAVID WATERSTON.

**CONTRIBUTION TO THE ENOEPHALIC ANATOMY OF THE**  
**(46) RACES.** First Paper — "Three Eskimo Brains from Smith's Sound." By EDWARD ANTHONY SPITZKA, M.D., *American Journal of Anatomy*, vol. ii. No. 1.

THIS paper includes measurements during life of the heads of the individuals whose brains are described, measurements of the skulls, and an elaborate description of the fissures and convolutions of the hemispheres in each case, and is prefaced by a very short account of the "ethnic traits and anthropological status of the Eskimo."

Two of the skulls were brachycephalic, and one dolichocephalic. The weights of the brains, and of others described by other writers, were rather above the average European—1457 gms. for the males, and 1375 gms. for the females, after preservation.

As a result of a detailed examination of the cortical surface in

each specimen, the writer finds "prevailing typical differences which distinguish these from the brains of whites," although he is unable to describe the distinction in so many words.

There is a marked tendency in all to transverse fissuration, even in the brain from the dolichocephalic skull, and the most striking characters are the "different relative topography and boundaries of the cuneus, the exposition of the insula, and the preponderating development of certain features upon the left, and of others on the right side." The brains, on the whole, are highly developed, and this account therefore adds another to those descriptions of the brains of aboriginal races which show that even in low races there may be good cerebral development.

DAVID WATERSTON.

### PHYSIOLOGY.

**EXPERIMENTS ON THE CONDUCTIVITY OF THE SPINAL**  
(47) **CORD RENDERED ANÆMIC BY COMPRESSION OF THE**  
**AORTA.** By MAX LOEWENTHAL, *Brain*, Autumn, 1902, p.  
274.

THE object of these experiments was to determine the effect which complete anæmia of the spinal cord has on its conductivity for centrifugal impulses, and at the same time to repeat some of the observations previously made by others on the reflexes, under the same conditions. Cats were used. The lower half of the cord was rendered temporarily anæmic by compressing the thoracic aorta. This was done by resecting part of the sixth or seventh rib on the left side, introducing the index finger into the pleural cavity, and compressing the aorta against the vertebral column between this finger and the thumb of the same hand placed outside. The animals were anæsthetised, and artificial respiration was kept up.

The left motor area was exposed, and the hind limb centre stimulated by a faradic current just strong enough to give a response. Then the aorta was compressed and the cortex stimulated simultaneously. At first the hind limbs responded much more vigorously and promptly than before compression, and with quickly increasing strength, until after about forty seconds the movements began to be weaker, and after eighty seconds they had ceased altogether.

The knee-jerks showed the same peculiarity as the cortical response; they attained their maximum after about forty seconds and had entirely disappeared in about eighty seconds.

In some cases, when "ischæmic paralysis" was commencing,

localised convulsions were observed in the posterior part of the animal's body, analogous to the general convulsions observed in animals bled to death. "They certainly originate in the grey matter of the spinal cord and therefore deserve the name of 'Spinal Fits.'"

On stimulating the sciatic nerve, the resulting reflex movements disappeared after from two to three minutes, but there was no initial increase, as in the case of the knee-jerk and cortical response. The same was observed on stimulating the posterior nerve roots of the lumbar enlargement, but "the weakest current applied as late as fifteen minutes after death at the insertion of the posterior roots gave a violent response." Here the stimulation was presumably applied to the tip of the posterior horn, the muscles on both sides responded, and this the author believes to be a genuine reflex and "not due to the anterior roots being stimulated by stray loops of current."

The author is of the opinion that the interruption in the conductivity takes place in the grey matter, at the synapses where one neurone is linked on to another, and not in the white matter as is supposed by Hering and others. This opinion he supports by very convincing arguments for which the original paper may be consulted.

He arrives at the following general conclusions :—

"1. The first effect of arrest of the circulation on the functions of the spinal cord is an increase in the readiness and strength of interneural transmission of nerve energy lasting about thirty seconds."

"2. After from one to two minutes, stimuli approaching the grey matter through peripheral nerves or the fibres of the white substance are not transmitted to the lower motor neuron."

"3. It is probable that every single neuron as a functional unit retains its vitality for a period exceeding fifteen minutes, but that the mechanism which links the separate neurons together is quickly destroyed by death."

"4. The existence of genuine spinal fits cannot be disputed."

SUTHERLAND SIMPSON.

## PSYCHOLOGY.

### ÜBER DIE BEZIEHUNGEN DER ENERGETIK ZUR SEELEN-

(48) THÄTIGKEIT. A. ADLER, *Neurol. Centralbl.*, Dec. 16, 1902, S. 1139.

THIS paper is a highly condensed, almost epigrammatic, statement of the writer's speculations regarding the physics of cerebral action. By the term *Energetik* he means the activity of the nerve

cells directed to the renewal of the material which maintains their tone. He starts with the proposition that the cortical cells answer every stimulus by a change in their molecular activity. Change in this activity is perceived as a sensation, and the strength of the sensation is dependent on the degree of change produced by the stimulus. By a process of elaboration this idea is brought to bear on the analysis of complex sensations. Wundt's law of sensation, mental functions such as association, judgment, emotion, and lastly free will, are sought to be explained on a similar basis. Whether the author is successful or not is decidedly doubtful, but the paper is so condensed and speculative that a short summary cannot do it full justice. It should be read in full. JAS. MIDDLEMASS.

**MENTAL GROWTH AND DECAY.** By E. C. SANFORD,  
(49) *Am. Jour. Psychol.*, July 1902, p. 246.

THIS is a psychologist's sketch of the course of mental development from the first beginnings of mind at, or before, birth, to the final failure and break-up of the powers in old age.

*Infancy* is characterised by the gradual development of voluntary motor power, and this is exemplified by a description of the acquisition of voluntary control of the arm and hand. Along with this there occurs an increase in the power of tactile discrimination, a growth and refinement really of a crop of tactual ideas.

With the end of his third year the baby passes into *Childhood*—the age of general mental adjustments, as babyhood was that of physical adjustments. Early childhood is a period of somewhat rapid growth and change. Mental development occurs under the guidance of several strong and important instincts: the instinct to investigate, to ask questions, to experiment; and especially to imitate and to play. The child lives the life of the senses, simpler motor activities, and imagination. His moral standards are borrowed from adults and often shifting. Later childhood, from nine to twelve or fourteen, is the school period *par excellence*, the time for learning things. It is the time for learning multiplication tables and whatever else comes only by drill and drudgery. It is the time for beginning practice upon musical instruments, for manual training and the like. In conduct and morals also it is a time for drill.

*Adolescence* extends from the close of the last period to that of physical maturity. It is a period not only of profound physical change, but of equally profound mental change. The reasoning powers develop rapidly and the eyes open to the larger problems of life. The most characteristic changes, however, are those which root in emotion. It is the time for love; for hero-worship; for awakening interest in moral and religious questions.

*Young Manhood* is characteristically the period for action. After forty a man becomes less and less aggressive, learns his limitations, and gradually converts his ambitions into ambitions for his children.

The *Period of the Elderly* extends from fifty-five or sixty to seventy. In it physical decline is unmistakable. Intellectual vigour may survive, but a man must take care of himself.

*Old Age*.—By seventy all the man's physical incapacities are emphasised. The bodily tissues show signs of degeneration. The nerve cells show many of the appearances that characterise fatigue in younger people. The mental marks are too great fixity of habit in thought, too little power of origination, and too little courage for new undertakings, a tendency to revert to the affairs of youth and early manhood, defective memory, defective powers of sustained effort.

W. B. DRUMMOND.

**FIXED VISUALISATION: THREE NEW FORMS.** By EDWARD (50) FRANKLIN BUCHNER, *Am. Jour. Psychol.*, July 1902, p. 355.

THIS is an account of a female subject, thirty-five years of age, who had the following visual forms:—(1) A number form, which appeared to the subject in a half fan-like radiation, extending forward and to the left of the mental point of regard. (2) A day form. This is a tri-dimensional form into which the days of the week are placed. (3) The month form. The months comprising the current calendar year are arranged by the subject into a form, which is distinct from the form for the days. These forms are illustrated by diagrams.

W. B. DRUMMOND.

## PATHOLOGY.

**BEITRÄGE ZUR PATHOLOGIE DER KLEINEN HIRNGEFÄSSE.**

(51) DEGENKOLB, *Allg. Zeitschr. für Psychiatrie*, Bd. 59, H. 5, S. 714.

A. On the occurrence of intra-adventitial cell-infiltration of the cortical vessels in diffuse lesions.

Only such cell-infiltrates are included as lie in part at least in the adventitial space, and consist of leucocytes or their derivatives, or plasma cells, but excluding mere increase in the number of nuclei of adventitial cells.

Intra-adventitial cell-accumulations in the near neighbourhood of small necrotic foci, neoplasms, miliary aneurysms or nodules of extreme endarteritis are excluded here also, as although leucocytes

occur in the adventitia they are not numerous enough in uncomplicated cases to constitute a regular infiltration. The occasional occurrence of leucocytes in hyaline fibroid degeneration (Robertson) and hyaline-sclerosis (Alzheimer) falls in the latter category also.

Practically a cell-infiltrate can be regarded as of inflammatory or irritative origin, when 20 true infiltration cells occur in a stretch of 0.3 mm. The occurrence of similar infiltrates in psychoses following trauma is so rare as to awaken the suspicion that the trauma acts only as an exciting cause for a latent pre-existing condition.

Intra-adventitial cell accumulations have been described in the following conditions:—

Infective brain affections, including non-suppurative encephalitis. It is noteworthy that in cases with bacteria in the brain fully developed infiltrates may be absent. They are constant in syphilis among chronic infections, but according to D. are not invariably confined to the grey matter.

Their occurrence in some idiot brains is probably referable to previous encephalitis.

In cortical affections due to pure toxin-action, cell-infiltrations are generally absent, *e.g.* post-diphtheritic neuroses; they may occur in very acute and severe toxæmias.

Acute phosphorus and alcohol poisoning also give rise to cell-infiltrates, while carbonic oxide and lead do not. They may occur in delirium tremens.

Apart from the above, general paralysis is the only diffuse cortical affection in which intra-adventitial infiltrates are constant. They seemed to be absent in one of Binswanger's early cases.

D. regards the attempt to deduce the infective or syphilitic origin of general paralysis from such consideration as futile, and believes that the cyto-biological character of these intra-adventitial cell-infiltrates will indicate the nature of the pathological process.

#### *B. On nuclear globules in intima-cells.*

Minute globules of a fatty nature attached to or half-imbedded in the nuclei of the intima-cells are never entirely absent. They are increased in conditions which produce general fatty degeneration and probably indicate fatty degeneration of the intima of the cortical vessels. Their physiological occurrence, according to D., indicates an unrecognised function of the intima-cells, that namely of transmitting nutriment to the underlying tissues.

J. A. MURRAY.

**NOTE ON CELL CHANGES IN A CASE OF COMPLETE COMPRESSION OF THE CORD.** By JOHN JENKS THOMAS, *Jour. of Nerv. and Ment. Dis.*, Oct. 1902, p. 599.

AN unmarried woman at age of 21 began to have weakness in the legs, which soon developed into complete sensory and motor paralysis, and persisted for thirteen years until death from exhaustion. The lesion was an endothelioma of the dura, which compressed practically the whole cord at the mid-dorsal region; only a few fibres in the periphery of the posterior columns apparently escaping. There was ascending and descending degeneration in the usual tracts. The neuroglia was greatly increased at the site of pressure, and the cord below was much atrophied. The legs were wasted, and there was marked contracture.

The lumbar enlargement was examined microscopically after hardening in formalin, embedding in paraffin, and staining by Nissl's methylene blue method. Some of the multipolar cells were normal to all appearance, a few showed wasting of the nucleus and diffuse irregular staining with methylene blue, while the remaining cells were definitely affected. These show no abnormality in the peripherally-placed chromatic granules, but there is a central chromatolysis and the axis cylinder hillocks show a similar diffuse granular appearance and are stained faintly blue. The nuclear membrane is indistinct or else irregular, and what appears to be a local heaping up of the chromatin was noted along the line of the nuclear membrane. The body of the nucleus was clear, and the nucleus is generally in its usual position in the cell. The nucleolus in some of these cells is swollen and vacuolated, and may be excentric in position. In many of these cells almost one-third of the whole area of the protoplasm does not stain even diffusely, and is evidently the site of accumulations of pigment which have a slightly yellow colour.

Dr Thomas argues that these changes indicate that loss of stimuli from the central motor neurones may cause definite lesions in the cell centres of the peripheral neurones, but inasmuch as many cells escape, the degenerative process is presumably a very slow or incomplete one.

R. A. FLEMING.

**SULLE MODIFICAZIONI DELLA PRESSIONE SUB-ARACH-  
(53) NOIDEA E DEI CARATTERI DEL LIQUIDO CEREBRO-  
SPINALE NELLA EPILESSIA SPERIMENTALE. A.  
D'ORMEA, *Riv. Speriment. di Freniatria*, 1902, f. 2-3, p. 49.**

**SUR L'INNERVATION MOTRICE DES VAISSEAUX DU  
(54) CERVEAU ET DE LA MOELLE. E. CAVAZZANI, *Archives  
ital. de Biologie*, 1902, T. 38, f. 1, p. 17.**

D'ORMEA's experiments were carried out upon dogs, narcotised by intravenous injection of full doses of morphia. The "sub-arachnoid" pressure was registered by Cavazzani's method, that is to say by means of a cannula passed into the sub-dural space through an opening made between the occipital bone and the atlas vertebra. Fits were induced either by direct electrical stimulation of the motor zone of the cortex or by intravenous injection of essence of absinthe. The author found that during the seizures the cerebrospinal fluid became turbid and of a reddish colour. These changes were evidently due to the presence of red blood corpuscles. The chief conclusion derived from his experiments is that during an epileptic fit the "sub-arachnoid" pressure almost constantly undergoes a sudden and considerable increase which reaches its height in the tonic phase and first part of the clonic period, after which it slowly diminishes with numerous and irregular oscillations. He confesses himself unable to interpret the significance of these results. He does not allude to Leonard Hill's observations upon the same subject. Anyone acquainted with the important work of the British physiologist would, however, at once attribute the increase of intracranial pressure observed in these experiments to a rise of intracranial venous pressure, following the rise of the general venous pressure caused by the convulsive movements. This being so, one would be disposed to pass over D'Ormea's observations as of little account, were it not that they formed the starting-point of an experimental research by a more experienced worker in the same very difficult field.

Cavazzani, in whose laboratory D'Ormea's observations were made, clearly recognised that the rise of intracranial pressure in these experiments might be due to elevation of the venous pressure, or to general energetic constriction of the arteries, but he had reasons for believing that these did not furnish the full explanation of the phenomenon. His own previous experimental observations had appeared to demonstrate that there is a vaso-dilator mechanism for the cerebral arteries which seems to predominate over a constrictor mechanism. He therefore thought it probable that an investigation of the causes of the rise of intracranial pressure during experimentally-produced epileptic fits would throw addi-



tional light upon this question of the innervation of the cerebral blood-vessels.

By observations upon dogs under the influence of morphia and curare, he determined that during fits induced by the action of absinthe, a well-marked elevation of the "sub-arachnoid" pressure takes place independently of muscular contractions and compression of veins. He then made another series of experiments with the purpose of ascertaining if the rise of intracranial pressure could be attributed to the general rise of arterial pressure that occurs during an epileptic fit. In these experiments he registered the general arterial pressure and the pressure in the circle of Willis. Summing up the results of these researches, he concludes that during fits induced by absinthe there is a lowering of the pressure in the circle of Willis which is independent of the conditions of the general blood pressure and accompanied by an augmentation of the "sub-arachnoid" pressure, i.e. of the volume of the brain and cord. He maintains that this phenomenon can only occur through an active dilatation of the cerebro-spinal vessels. The rapid and energetic way in which the dilatation takes place justifies the belief that it depends upon an excitement of special nerve-centres upon which absinthe has an irritant action. These experimental observations thus furnish, in his opinion, new evidence in support of the existence of vaso-dilator centres for the vessels of the brain and spinal cord. W. FORD ROBERTSON.

**PRINCIPI TOSIICI DEGLI ASPERGILLI FUMIGATUS E FLAVES-**

(55) **CENS, E LORO RAPPORTI COLLA PELLAGRA.** CARLO CENI e CARLO BESTA, *Riv. Speriment. di Freniatria*, 1902, f. 4, p. 528.

**L' AZIONE DEL SUOCO GASTRO-ENTERICO SULLE SPORE**

(56) **ASPERGILLARI IN RAPPORTO COLLA GENESI DELLA PELLAGRA.** CARLO CENI, *Riv. Speriment. di Freniatria*, 1902, f. 4, p. 688.

ALTHOUGH pellagra is not a disease of this country, there are reasons why the recently elucidated facts regarding its pathology (see p. 37) should be of more than ordinary general scientific interest. Among these reasons there is the circumstance that, thanks to Ceni's most brilliant researches, pellagra now presents one of the best examples of a "nervous disease" that has been clearly traced to the action of a micro-organism. With so large a field in neurology and psychiatry still waiting for the similar application of bacteriological methods, every detail regarding the pathogenesis of pellagra may offer valuable suggestion for new researches.

Ceni and Besta have endeavoured to extract the toxic principles from the spores of the aspergilli which cause pellagra, and to compare their action with that of the spores themselves. From *aspergillus fumigatus* at least they have succeeded in obtaining an extract which when injected into the peritoneal cavity of dogs and rabbits produces the same effects as the spores in conditions under which they cannot proliferate. They sum up the conclusions warranted by their observations as follows:—(1) Recent growths of *aspergillus fumigatus* after prolonged digestion with alcohol or ether yield substances endowed with very virulent toxic action and having specific characters. Recent growths of *aspergillus flavescens* yield much less active toxic substances, and only with ether. (2) The toxic principles are contained exclusively in the spores of these hyphomycetes. No toxins can be extracted from the mycelium. (3) In the case of *aspergillus fumigatus*, the toxic properties are in part related to the quantity of the spores, but in greater measure to special characters of sporification. Generally speaking the toxic power is inversely proportional to the development of mycelium and directly proportional to the maturity of the spores. The nutritive medium upon which the germs develop is not of special importance. (4) The toxic power of the extract of *aspergillus fumigatus* dissolved in water, and left alone, diminishes somewhat rapidly. On the other hand it is not affected by prolonged boiling or by the action of alcohol.

Ceni has himself investigated the action of the gastro-intestinal secretion upon these aspergilli, and has succeeded in throwing important additional light upon the pathogenesis of pellagra. His object was to ascertain why it is that the spores of the aspergilli when injected into the vessels or tissues give rise to pseudo-tuberculosis only, whilst if made to enter by way of the alimentary tract they produce special neuro-toxic symptoms similar to those of pellagra. He has found that the gastric and enteric secretions, but especially the latter, have a powerful inhibitory action upon the growth of these organisms, and also possess sporicidal properties. Spores subjected for a time to the action of the enteric secretion of the dog, produced, when injected into guinea-pigs, an intoxication quite different from that which ordinarily results from the injection of the spores. In cases of pellagra, the spores having been introduced into the alimentary tract are there evidently inhibited in their growth, but they preserve their toxic properties for a considerable length of time and thus give rise to a true intoxication, instead of to the phenomena of the common aspergillosis. Many of the spores pass into the circulation, lodge in various tissues and organs, and remaining there as spores determine the phenomena of pellagrous intoxication.

W. FORD ROBERTSON.

**THE PERIPHERAL THEORY OF NERVE REGENERATION WITH  
(57) SPECIAL REFERENCE TO PERIPHERAL NEURITIS.**

Read before the Edinburgh Med.-Chir. Soc., July 2, 1902, by  
ROBERT A. FLEMING, *Scottish Med. and Surg. Journal*, Sept.  
1902.

**REFERENCE** is first made to the old or central and the new or peripheral theories of regeneration.

According to the old or central theory the original axis cylinders in the central end of a divided nerve either grow downwards themselves or else they divide, and the young axis cylinders so formed pass downwards inside the old parent neurilemma sheaths. Probably in the peripheral segment when regeneration has occurred in this way, the existing neurilemma nuclei form new neurilemma sheaths. Bowlby, MacCormac, Kennedy, and other surgeons found that after secondary suture of a nerve divided for months or even two or three years, sensation sometimes returned in the realm of the sutured nerve in one or two days. The explanations offered by the central theorists that some nerve fibres do not degenerate in the peripheral segment or that anastomosis might occur, are not satisfactory and can not be demonstrated.

According to the new or peripheral theory formulated by Von Büngner in 1891 supported by Eichhorst, Neumann, Tizzoni, Bethe, and among embryologists by Beard and others, the peripheral segment of a divided nerve completely degenerates, beginning in a few hours and being nearly complete in three or four weeks. Among the phagocytes the neurilemma nuclei which proliferate and form cells with large oval nuclei and little protoplasm aid the leucocytes and connective tissue cells, but soon they take on the function of neuroblasts, become arranged in rows, and from their protoplasm both young axis cylinders and eventually the surrounding myelin sheaths develop. These young axis cylinders unite end to end and remain as imperfect chains until united to the central end of the nerve. The steps in this process of neuroblastic regeneration have been recently demonstrated by Kennedy in 1898 and more lately by Messrs Ballance and Purves Stewart.

The author, while holding the peripheral or neuroblastic theory of regeneration, believes that there is also a down-growth of young axis cylinders which have formed by division from the central end of the divided nerve, and a few out of a large number of experimental sections and ligations of rabbit sciatics are described in support of his views.

In peripheral neuritis the toxin acts not merely on the neurones with their cell centres, but also in many cases on the blood-vessels

belonging to the affected nerves and muscles and other tissues in the realms supplied by these nerves. These vascular changes with the associated exudations and the marked degeneration of the fine medullated nerve fibres are fully described in an article in *Brain* published in 1897. Many of the larger nerve fibres also degenerate in the affected nerves in a marked case of peripheral neuritis, but invariably the fine fibres are most severely and also are the first to be affected. If transverse sections of any nerve are studied, these very fine medullated fibres are found in small groups, and it is therefore in the position of these groups that regeneration should be first looked for. Four fatal cases of alcoholic peripheral neuritis are described, in all of which regeneration of neuroblastic origin was present in the affected nerves, and it was extremely marked in one case. The rapid onset of the nerve affection in one of the cases with death within three weeks and in another the small number of degenerated fibres in the affected nerves rendered it unlikely that regeneration would be present in more than its earliest stages.

Similar regeneration has been noted in a number of other fatal cases of alcoholic neuritis which have not yet been published.

AUTHOR'S ABSTRACT.

**GIEBT ES EINE AUTOGENETISCHE REGENERATION DER  
(58) NERVENFASERN? EIN BEITRAG ZUR LEHRE VOM  
NEURON. Von EGMONT MÜNZER, *Neurol. Centralbl.*, Dec. 1,  
1902.**

THE paper is an attack on Bethe's views with regard to neuroblastic regeneration, and a defence of the neurone theory, which the author considers is endangered by the acceptance of the peripheral theory of regeneration. After defining Bethe's position, the author gives details of a small series (10) experiments on rabbits in which the sciatic was divided, a small portion removed (1.5 to 1.8 cm.), and the animals killed at various dates from 32 to 157 days. Münzer finds that a small "knotten" or nodule had formed at the site of the section to which the peripheral end of the nerve was found adherent. This, stained with osmic acid, was of a pale brownish colour, and was composed of very fine young nerve fibres running in all directions and extending downwards around and into the peripheral segment of the nerve. He considers that they originate from the central or proximal segment. This appearance is best marked in those rabbits allowed to survive after operation for at least 54 days.

Münzer also assails Bethe's statement that when a peripheral

nerve is cut and reunion prevented, and after regeneration has occurred in the peripheral segment (according to the peripheral theory) if the regenerated peripheral part be again divided the central part will not degenerate while the peripheral does.

He not merely denies the peripheral theory of regeneration and attacks the conclusions arrived at, but does not offer any experimental evidence in support of his own views.

The paper is a defence of the old central theory of regeneration, but the results are based on an altogether inadequate number of experiments.

ROBERT A. FLEMING.

**PATHOGÉNIE DU TABES DORSAL.** NAGEOTTE, *La Presse Médicale*, (59) Dec. 10, 1902.

IN this paper on the pathogenesis of Tabes, the author defines Tabes as a disease which is characterised anatomically by an inflammatory lesion, which attacks a certain number of sensory or motor nerve roots at their exit from the sub-arachnoid space, and which is due to a syphilitic meningitis. This meningitis by itself is not sufficient to produce tabes, because a meningitis may be present and still no symptoms of tabes appear. In all his cases of tabes he has found what he has called a transverse interstitial neuritis of the nerve root, which has spread inwards from the meningitis, and has affected the intrafascicular connective tissue. It is true that this intra-fascicular connective tissue may be affected in cases of non-syphilitic meningitis, and may give rise to symptoms analogous to those seen in tabes, but he insists that the progressive character of tabes is due to the syphilitic origin of the meningitis. He mentions that the intra-fascicular tissue is derived from the pia mater, although its texture is much more coarse and that the perifascicular tissues are derived from the subarachnoid tissue and the dura mater.

The nervous structures which are principally attacked are the posterior nerve roots and the posterior columns throughout their length. In an early stage the lesion is seen in the intra-medullary portion of the root fibres, while the extra-medullary portion may be intact, and it is probable that the distal portion of the neuron is first attacked, and that the disease progresses slowly toward the trophic ganglia. The short and medium lengthed fibres are more liable to be injured than the longer fibres.

The process consists of a progressive atrophy of each fibre, characterised by a thinning and disappearance of the myelin sheath, while the axis cylinder may persist for a long time. This process differs essentially from a Wallerian degeneration. The distribution of the lesions varies greatly in different cases; some-

times all the posterior roots of the cord are affected ; at other times the roots are attacked quite irregularly, and even certain fasciculi of the same root may escape while others are attacked. Later, most of the endogenous systems of the posterior columns become affected, but the system of short fibres, which borders on the grey matter, may persist indefinitely. Atrophy is also found in the antero-lateral tracts and in the crossed pyramidal tracts, and, in fact, the whole cord is diminished in volume. This is attributed by the author to a diffuse meningo-myelitis.

The meningitis may also attack the anterior roots, and this is especially frequent in the lumbo-sacral region. With regard to the parenchymatous lesions found in tabes, the author says that they do not possess any distinctive pathological characters.

The connective tissues present the histological appearances of a subacute or chronic inflammation, and correspond to those produced by syphilis in other organs. The pia mater is thickened and infiltrated by lymphocytes ; some plasma cells and a few polynuclear cells may also be present. The vessels are always altered, the veins being particularly attacked. They are infiltrated by lymphocytes which may be collected into nodules and which may distort the outline of the vessel.

In the arteries the outer coat is often affected alone, or it may be associated with an endarteritis, more or less rich in nuclei.

The alterations in the capillaries are discrete and scattered ; rarely the lesions of the capillaries may form a focus of myelitis.

The inflammatory lesion of the connective tissues is frequently most marked in the dorsal region, but it may sometimes be traced to the base of the brain and even to the cortex.

With regard to the time at which this meningitis appears, the author says that lumbo-sacral puncture has shown that there is always a marked lymphocytosis in the spinal fluid before there are any signs to show that the posterior roots have been attacked. This lymphocytosis is characteristic of all syphilitic affections of the central nervous system in which the meninges are involved, and the cytological examination proves therefore that the meningitis precedes the parenchymatous changes.

R. G. Rows.

#### **ZUR PATHOLOGIE DER PROGRESSIVEN PARALYSE. C.**

(60) FÜRSTNER, *Monatsschr. f. Psychiatr. u. Neurol.*, Nov. 1902, S. 409.

In a lengthy article Fürstner deals with the subject of general paralysis both from a clinical and from a morbid anatomy point of view. He brings forward arguments in support of Mendel's

assertion that the classical form of the disease in which the cases exhibit maniacal or hypochondriacal tendencies, exalted ideas, together with profound bodily disturbances, is becoming rarer; and is being replaced by the demented form where the principal symptom is a simple progressive failure of the intellect. On the whole, he favours the view that the disease is on the increase, but is careful to point out that too much reliance should not be placed on asylum statistics. During the last ten years 280 males and 72 females have been admitted to the Strassburger Clinic, and in the recent years the numbers admitted show a decided and gradual increase; the percentage of males to females has been 4 to 1; female cases have been surprisingly rare. This increase is attributed to drink, venery, pregnancies and penury; he does not think that youth can be said with certainty to supply an increase on account of the difficulty of arriving at an accurate diagnosis.

With regard to the duration of the disease, that seems to be shorter than formerly; about half of his cases die at the end of the second year, and a goodly percentage earlier than this; according to Gross the disease runs a quicker course in the female than in the male.

Post-mortem examinations do not reveal such gross changes as formerly. In 97 autopsies, great atrophy of the brain was present 9 times; hæmatoma of the dura (subdural false membrane) 6 times; hydrocephalus with ependymal growths 20 times; in 124 autopsies hæmatoma was present only 5 times (Gross). This rarity of subdural false membrane is ascribed to the shorter duration of the disease. Statistics lent by Schüle are quoted, viz., in 52 cases atrophy of the brain, turbid fluid and thickening of the pia were present in the majority of the cases; varying degrees of internal hydrocephalus were present 29 times. Regarding these facts Fürstner thinks that they may be due to the prevalence of the demented form of the disease and advises special attention to be paid to post-mortem findings in cases where the clinical symptoms are fully noted. His belief is that the lesions found after death on present-day general paralytics are presumably insufficient to account for the grosser characteristic clinical symptoms of the classical form of the disease. He admits that these cases are still occasionally met with, but that they are rare; and states that if the cases which show a short initial period of excitement followed by a simple progressive intellectual decline are granted to belong to the demented or taboparalytic form, then this type undoubtedly prevails. With regard to the morbid changes in the spinal cord, he considers that these may begin either in the posterior or in the lateral columns and spread from the one to the other; a comparatively small percentage of the cases show lesions limited to the posterior columns (12 per cent.

gross). He suggests that the term "Taboparalysis" should be limited to this variety.

Syphilis is put forward as the prime factor of causation, but on this question statistics are unreliable on account of the want of unanimity on the part of observers as to the changes which might or might not be syphilitic. Antispecific remedies, in contra-distinction to the results of most other investigators, have given him encouraging results. No conclusions are arrived at as to the localisation of the disease in the brain, and he says that no reliable guides have been obtained from microscopical examinations.

The author deals at some length with the use of the term "pseudo-paralysis"; as it is used at present, about 20 per cent of the cases come under it. As a rule he suggests that it would be better to use a descriptive term like paralysis with sensory, motor or aphasic symptoms; or paralysis with changes in the basal ganglia or other parts as the cases showed themselves. The "pseudo-paralysis" following alcohol, lead and syphilis he strongly objects to, as these cases may not run the course of general paralysis at all, and in genuine cases they are regarded as ætiological factors. Stress is laid on the following points as diagnostic of such cases, viz., presence of patellar reflex, hyperæsthesia, and paræsthesia of the extremities, absence of fixed pupils and the psychical symptoms peculiar to general paralysis.

He recommends that the term "pseudo-paralysis" be used for a class of case that at first shows undoubted signs of general paralysis, and which ultimately gets well. He mentions eight cases of this nature, all of which showed a hereditary predisposition combined with mental and bodily overwork and sexual excess. The symptoms were at the beginning, a period of short maniacal excitement with exalted ideas, tremor of the facial muscles, pupillary differences, irritability of the patellar reflex, and a certain degree of aphasia. All recovered and have been following their employment for periods of from seven to eleven years. Such cases, he believes, can only be explained on the ground that the morbid changes present in the brain cells during the attack disappear, and these affected cells recover their normal function so that no trace of the illness remains.

W. MAULE SMITH.

### CLINICAL NEUROLOGY.

**DES POLYNÉVRITES CHEZ LES ENFANTS.** MAURICE PERRIN,  
(61) *Archives de Méd. des Enfants*, Tome v. No. 12, Dec. 1902,  
p. 725.

In a careful paper, with numerous references to literature, M. Perrin passes in review the causes symptoms, diagnosis and prog-



nosis of multiple neuritis in children. Save in the frequency of its occurrence, there seems to be little difference in the disease at different ages.

If we except diphtheritic paralysis, affections of the peripheral nerves are much less common in childhood than they are in later life. In early infancy, indeed, multiple neuritis is extremely rare. As age advances, however, it increases in frequency of occurrence, and it is not uncommon in adolescents. The fact that young children are less prone to disease of the peripheral nerves is interesting in view of their special liability to lesions of the cells in the anterior horns. When multiple neuritis does occur in childhood, it shows either that there is a strong hereditary predisposition present, or that the morbid influence at work is one which possesses a very special affinity for the peripheral nerves.

JOHN THOMSON.

**EARLY MANIFESTATIONS OF TABES.** By WILFRED HARRIS,  
(62) *Practitioner*, October 1902, p. 395.

In an analysis of 46 early cases of the disease, stress is laid on the frequency with which brisk or exaggerated deep reflexes are met with, cases of undoubted tabes and not general paralysis or ataxic paraplegia as they are frequently called simply on account of the presence of the knee-jerks. Erb is quoted in support of the diagnosis of tabes in many atypical cases, which may never develop fully, but on the other hand may at some time run a rapid and severe course. Of the 46 cases, the initial symptom was lightning pains in 45·6%, while no less than 16% commenced with definite gastric symptoms. The protean character of the disease is shown by the numerous and diversified symptoms first complained of. Two cases complained of pain round the chest as the first symptom, one began with bladder trouble, another had spontaneous fracture in both legs, two had various paræsthesiæ, one had vertigo, another optic atrophy, one came for herpes ophthalmicus, another with fifth nerve neuralgia, and in three the signs of the disease were found accidentally, the patient having come for trifling cough or dyspepsia. In 48% of the cases the knee-jerks were brisk on both sides, but attention should be also directed in every case to the Achilles-jerk, the biceps and triceps jerks and the jaw-jerk, as not unfrequently some discrepancy will be found on the two sides, even when both knee-jerks are brisk.

In five cases, or 11%, gastric crises were the initial symptom, and in two of the cases whose crises were severe, with great pain, epileptic fits occurred during the crises. In view of the irritable state of the nerve centres in visceral crises as shown by the exces-

sive secretion of their mucous membranes, the theory is advanced that the pathology of these various visceral crises depends on paroxysmal epileptiform discharges in their nerve centres produced by irritation of these centres from commencing neighbouring degeneration, or due to reflex stimuli reaching these centres from diseased nerves in the periphery. It is further noted that chronic indigestion for twelve months or more may precede the onset of the gastric crises, suggesting a peripheral cause in the stomach reflexly aiding in the development of the attacks. Several cases with ocular paralyses are described, amongst them being two whose first symptom was sudden complete third nerve paralysis. In five of the 46 cases, or 11%, diplopia or ocular palsy was the initial symptom.

*The pupils.*—In only 4 cases were the pupil reactions to light normal; in the remainder, or 91·3%, there being diminution or loss of light reaction in one or both eyes. In one of the cases with normal pupil reaction described, the case closely resembled in many points a case of sensory peripheral neuritis, with hyperæsthesia of the fingers and toes, and thin, sweaty skin. The conclusion is arrived at that reflex iridoplegia, though strongly suggestive of syphilis, may certainly occur in its absence; uniocular Argyll-Robertson pupil was observed in five of the cases, in one case the loss of light reaction being accompanied by loss of reaction to accommodation and also paralysis of accommodation itself, while in the other eye the pupil and ciliary muscle were normal, but there was complete paralysis of the external rectus. Other cranial nerve affections in tabes are mentioned, notably one case with complete paralysis of the sensory portion of the right fifth nerve, the initial symptom being severe trigeminal neuralgia. Stress is laid on the importance of analgesia on the outer side of the leg and on the thorax as early signs in the diagnosis of tabes, occurring sometimes before loss of the knee-jerk or of the pupil reflex to light. The diagnosis from general paralysis, syringomyelia peripheral neuritis and cerebro-spinal syphilis is discussed, and a short abstract given of each of the 46 cases.

AUTHOR'S ABSTRACT.

**REMARKS ON THE EARLY RECOGNITION OF TABES DOR-**  
(63) **SALIS.** By R. T. WILLIAMSON, *Medical Chronicle*, Aug. 1902.

THE author, while insisting that the diagnosis of tabes should never be based on one symptom, emphasises the value of a combination of two or more of the following symptoms in the early diagnosis of tabes:—

(1) Shooting pains; (2) absence of knee-jerk; (3) absence of the tendo-Achillis reflex—in the very rare cases in which the knee-

jerk is present in the early stage, the tendo-Achillis reflex may be lost (two cases cited); (4) the Argyll-Robertson pupil—not invariably present; (5) zones of diminished (not lost) cutaneous tactile sensation on the trunk, especially about the level of the fourth intercostal space, corresponding to the distribution of spinal nerve-roots—often absent in cases of early tabes with optic atrophy (present in two out of five cases without optic atrophy, in four out of eleven cases with optic atrophy); (6) loss of muscular tone (muscular hypotonus)—especially as tested by the height of elevation of the fully extended leg in the recumbent position, an angle of 65 to 75 degrees with the horizontal plane being rarely exceeded by a non-tabetic person (of fifty such persons only two exceeded 75 degrees), while in tabes, even at the early stage, the leg can often be raised to an angle of 80 to 100 degrees (seen in eighteen out of nineteen cases of tabes, mostly at the early stage); muscular hypotonus, although not a constant or pathognomonic sign, is of special value in early cases of tabes with optic atrophy, in which other symptoms are often very few; (7) ataxia or unsteadiness of gait (usually a later sign); (8) primary optic atrophy—may be the only symptom, and is generally a very early one; vision slowly diminishes with the progressive atrophy and ultimately there may be total blindness; concentric restriction of the visual field or sector-form restriction, but central scotomata exceedingly rare (in contrast with disseminated sclerosis).

Brief notes of fourteen cases of early tabes are appended.

ASHLEY W. MACKINTOSH.

**ANALYSIS OF 155 CASES OF TABES.** By BYROM BRAMWELL,  
(64) *Brain*, Spring, 1902, p. 19.

THE results of a careful and exhaustive analysis agree in the main with those of Leimbach, Bonar, Thomas, and Riley. Bramwell found a history of some form of venereal disease in 76·5%, but at the same time records his conviction that some of the cases who denied syphilis had certainly not suffered from the disease. In one case the tabes seemed to be due to diphtheria. He suggests that toxins other than that of syphilis may produce or predispose to the development of tabes. The majority of the cases developed from ten to twenty years after the syphilitic infection. In some cases sexual excess or marriage aggravated or accelerated the disease, and also occasionally alcoholic excess. Vulnerability of the nervous system from inherited constitutional weakness of nerve tissue may be a factor. Far the most frequent of all the symptoms was lightning pains, occurring as the initial symptom in 52·9%. Next in order of frequency as the first symptom of the

disease came diplopia and ataxy, in each 8·6%. Following these in diminishing frequency came loss of vision, bladder trouble, gastric crisis, and numbness of the feet. Lightning pains occurred at some time during the disease in no less than 95·4%, and in no case were they present in the arms or trunk alone, the legs always being involved, though occasionally in less degree. Anæsthesia in the legs was found in 59·6%, and on the thorax in 53·3, while thoracic analgesia was found in 75·6% of those tested, and ulnar analgesia in 48·3%. It is interesting to note that one case had complete left hemianæsthesia. Of the deep reflexes the knee-jerks were absent in 81·9%, and increased on both sides in 3%. The Achilles-jerk was absent in the same proportion as the knee-jerk, while in a very few the Achilles and knee-jerks did not correspond, one being lost while the other was retained or exaggerated.

The deep reflexes in the arms were absent in 50·9%, and exaggerated in 3%, but unfortunately no separate figures are given for the biceps and triceps jerks. The jaw-jerk was lost in 18%. In those cases in which the cremasteric reflex is affected, the sexual reflex is much more frequently affected than not. The *pupils* were contracted, less than 3 mm. in two-thirds of the cases, while the light reaction was completely or nearly lost in one or both eyes in 83·4%. In 16·5% the pupils reacted briskly to light. The complete Argyll-Robertson phenomenon, loss to light, but brisk to accommodation, was present in one or both eyes in 74·4% of the cases when first seen. Bramwell's results agree with Bonar's in the presence of the Argyll-Robertson pupil in the majority of those cases of tabes in which syphilis was denied, as well as in those in which the infection was admitted. *Optic atrophy* was present in 21%, being the initial symptom in nearly 6%. In several the tabetic symptoms abated after the development of the atrophy. *Gastric crises* occurred in 10%, 4% being in the pre-ataxic stage of the disease. Three of the cases had no stomach pain though the gastric crises were well marked. In another, irregular dyspepsia and vomiting was present for two years, perhaps an irregular form of crisis. In 2% laryngeal crises occurred, all in the ataxic stage. Charcot's joint disease was found in 6·4%, in one case the jaw being affected. In 49% there was considerable bodily wasting. The vascular system was affected in several, aortic aneurysm being present in 3 cases, and aortic regurgitation in 5 cases, while of 25 cases examined, the pulse average rate was above 85 per min. in 9 cases.

Of 30 fatal cases the average duration of the disease was 8·3 years, varying from twenty-two years to eighteen months in two cases.

WILFRED HARRIS.

**TABES DORSALIS ET AORTITE.** Par PIER. F. ARULLANI, *Revue*  
(65) *Neurologique*, Oct. 30, 1902, p. 970.

ATTENTION is directed to the importance of careful examination of the heart and vascular system in tabetics. Of 68 cases 11 were found to have aortic insufficiency, and 2 had aneurysm, while 29 others showed more or less marked signs of aortitis. The most obvious fact to be noticed is the tachycardia, 90 to 100 per min., and dulness to the right of the sternum indicating dilatation of the aorta. The heart is frequently dilated towards the left, while the aortic sounds are often modified, a loud second sound, or a diastolic bruit to be heard after exertion. These signs are probably due to myocardial disease. The aortic insufficiency is nearly always relative, due to dilatation, and the bruit may be present only after exertion. The patient must therefore be examined not only in the recumbent posture, but also when fatigued. Seventy-seven per cent. of his cases had had syphilis, but other toxins as alcohol, malaria, lead and small-pox are suggested as causes besides syphilis, not only of the aortitis but also of tabes.

WILFRED HARRIS.

**REMARKS ON ACUTE MYELITIS AND A REPORT OF A CASE**  
(66) **OF TUBERCULOUS MENINGO-MYELITIS.** By JOSEPH  
COLLINS, M.D., *Journ. of Nerv. and Ment. Dis.*, Dec. 1902.

THE author first comments upon the rarity of true acute myelitis and points out that the only test of its presence in any given case depends upon the existence of changes in the circulation typical of inflammation. He then gives somewhat meagre details of a case of meningitis of the cord and base of the brain with much softening of the spinal cord. The illness began with acute inflammation of several joints and the development of extreme wasting and rigidity of the lower extremities, severe bedsores, and incontinence of urine. About this time the patient gave birth to a healthy child, labour being absolutely painless. Some weeks later she developed signs of septicæmia and died three months after the onset of the joint symptoms.

Post-mortem the theca was found to contain pus below the second lumbar vertebra and greenish lymph above this, which extended also over the base of the brain. The diagnosis of the tuberculous nature of the affection rests upon the discovery of the bacillus of tuberculosis in one section from the dorsal region, no tuberculosis was found in any other part of the body. The cord showed chromatolysis and plasmolysis of cells and inflammatory

changes about the vessels. In concluding the author expresses the opinion that the initial joint symptoms were not rheumatic and questions whether the cord changes were truly inflammatory or due to nutritional defect resulting from the alteration in the blood supply produced by the meningitis.

H. DOUGLAS SINGER.

**REPORT OF A TRANSVERSE LESION OF THE MID-THORACIC (67) SEGMENTS LEAVING INTACT THE POSTERIOR COLUMNS, AND CAUSING SYRINGOMYELIC DISSOCIATION.** By ADOLF MEYER, *Journ. Nerv. and. Ment Dis.*, Dec. 1902, p. 715.

THE interest of this case lies in its bearing on sensory localisation in the spinal cord. The chief points of clinical importance—existing for probably two years before death—were:—practically complete motor paralysis below the thorax, with exaggerated knee-jerks and loss of control over the sphincters; no obvious atrophy; occasional involuntary pulling up of the thighs, at times accompanied by shooting pain; girdle feeling (occasional) at the level of the umbilicus; tactile sensibility, localisation, pressure sense, tickle sense and sense of position normal; analgesia and thermo-anæsthesia below the sixth rib on the right side and the fifth rib on the left side; a zone of slight thermo-hyperæsthesia over the fourth rib and a zone of tactile hyperæsthesia, about 8 cm. wide, extending on the right side downwards from the upper border of the sixth rib and on the left side from the upper border of the fifth rib.

The autopsy disclosed a myelitic destruction of practically the entire cross-section of the fourth to the sixth dorsal segment, leaving intact only the dorsal two-thirds of the posterior columns and a few pyramidal fibres. (Four figures are given, showing the extent of the lesion and the secondary degenerations at different levels of the cord.)

ASHLEY W. MACKINTOSH.

**SPINAL-CORD TUMORS.** J. COLLINS, *Medical Record*, Dec. 6, (68) 1902.

THE author discusses the symptomatology of spinal tumours and the diagnosis between the meningeal and intra-medullary forms.

Pain is rarely an initial symptom in intra-medullary spinal tumour, but in spinal meningeal tumours it is of great severity and is almost invariably an early symptom. There is danger of localising a spinal tumour one segment too low. The con-

sensus of opinion appears to be, that the location of any tumour in the cord is from two to four inches above the uppermost limits of the anæsthesia, and more often the latter figure than the former. The importance of spinal sensitiveness, as an aid to the localisation of the tumour, is very considerable, especially if it coincides with the region which the sensory and motor symptoms indicate to be the seat of the disease.

The wide-spread area of pain in spinal tumour is confusing; it is not often that the pain is confined to one or two nerve roots.

In 70 cases analysed by Collins the tumour was in the dorsal region in 35, in the cervical region in 15, in the lumbar and sacral region in 13, widely distributed in 6.

Collins records three cases of spinal tumour.

In Case i. the important symptoms were: (1) paroxysmal pains, chiefly in epigastric and umbilical regions; (2) motor weakness with spastic condition, beginning in one leg, gradually increasing and affecting the other leg afterwards; (3) Brown-Séquard's syndrome; (4) the gradually progressive course of the disease.

The growth was diagnosed as extra-medullary (meningeal) from the following points in the symptomatology: (1) the pain preceded the motor symptoms for one or two years; (2) the motor symptoms came on gradually; they were first unilateral and were not accompanied by atrophy; (3) Brown-Séquard syndrome was present.

Operation was performed by S. Lloyd. The laminæ of the 5th, 6th, 7th and 8th dorsal vertebræ were removed and a tumour found firmly attached to the inner surface of the dura mater. It was removed, and microscopically it "appeared to be sarcoma." Owing to the long duration of the compression of the cord, only slight improvement followed.

Case ii. was diagnosed during life as myelitis, following pneumonia; but the autopsy revealed a tumour (sarcoma) between the bodies of the vertebræ and the dura. The growth extended from between the 3rd and 4th cervical to the level of the first dorsal vertebræ. The tumour could have been removed, but the nature of the growth would have precluded any permanent benefit from operation.

In Case iii. paraplegia developed rather suddenly. There was localised pain in the back, increased greatly by movements of the body, anæsthesia of the legs and lower part of the abdomen, paralysis of bladder and rectum and a large sacral bed-sore. The autopsy revealed a sarcoma of the dura mater from the 9th to the 12th dorsal vertebræ.

Collins adds to his paper abstracts of the records of 70 cases of spinal tumour published during the last six years. Of these cases 30 were operated upon: the result was successful in 12

instances, partially successful in 8, and unsuccessful in 10 cases. Amongst the cases operated upon there were 6 cases of fibroma, 12 of sarcoma, 3 of endothelioma, and 1 of myolipoma. All of the cases of fibroma recovered except one, in which death occurred from sepsis.

Collins estimates that 44 out of the 70 cases were suitable for surgical treatment. He thinks that 50 per cent. of spinal tumours are suitable for operation, and that one-third to one-half of the cases are likely to derive benefit by operation. When the operation can be performed without the development of sepsis, the percentage of recoveries will be greater. The importance of early operation, before the cord changes have become too advanced, is pointed out; also the danger of delaying operation for the purpose of trying the action of iodides is emphasised, since gumma of the cord rarely produces the symptoms of spinal tumour. R. T. WILLIAMSON.

**RUCKENMARKSTUMOR MIT ERFOLG EXSTIRPIERT.** Von Prof.

(69) Dr S. E. HENSCHEN und Prof. Dr K. G. LENNANDER, *Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie*, Bd. x, H. 5, 1902, S. 673.

THIS paper contains a most elaborate history of a case of spinal tumour which was successfully removed by operation.

The following are some of the more important details of the case:—

The patient was a well developed, muscular man, aged fifty. In January 1889 (?), he noticed that he did not feel cold with his left leg, although sensations of touch and warmth were quite well felt. In February 1898, he had some pain in the right shoulder and upper arm, but as he was subject to rheumatism he attached no importance to this. In the middle of the summer of 1898, he had a peculiar feeling in the left groin and region of the left hip, a feeling like a belt over the upper part of the abdomen, and formication in the left leg and both hands. In August he had some subjective sensations in the left leg and difficulty in passing water, also some weakness in the right hand and right leg. In September he noticed that he did not feel the ground distinctly with his left leg. In October he had cramp-like twitchings in the right leg and hand. On a few occasions during November and December, he suffered from a very severe pain in the neck over the sixth cervical vertebra and a little to the right side of it. The pain lasted about half an hour at a time and was so severe as to almost make him weep.

He was admitted to hospital on December 15, 1899. On ad-



mission his most important symptoms were:—Weakness in all four limbs, especially the right arm and leg. The rhomboids, spinati, serratus magnus, pectorals, latissimus dorsi, deltoid, biceps, coraco and brachialis were unaffected; with the exception of these muscles all those of both upper limbs were paralysed to a greater or less degree, the paralysis being much more pronounced on the right than on the left side. The right triceps was the only muscle which is distinctly stated to have been atrophic. "All the muscles respond to Faradism." The left pupil was rather larger than the right. Tactile sensation was completely lost in the left and diminished in the right leg, and on the trunk as high as the navel, as well as on the ulnar surfaces of the arms. There was analgesia both to pain and temperature in the left leg and over the left side of the trunk. The knee-jerks were increased right > left; ankle-clonus was present on both sides, right > left. The triceps-jerks were not obtained; the biceps-jerks were present. The plantar reflexes were diminished. There was some difficulty in passing water. He had no pain, nor was there any tenderness on pressure over the vertebræ. There were no signs of disease in any of the other organs.

After his admission to the hospital his symptoms continued to progress, the right leg becoming completely paralysed. The anæsthesia and analgesia spread up to the second rib. The muscular sense was quite lost in the right leg, although unaffected in the left.

On January 6th, there was tenderness on pressure over the sixth cervical spine and to the right of it. On January 13th, the patient could only sit up in bed with difficulty. On January 28th, he could scarcely turn in bed.

On February 8th, he was operated upon by Professor Lennander. A spindle-shaped, hard tumour, 3·8 cm. in length, was found under the sixth cervical vertebra, between the pia and arachnoid and pressing on the right side of the cord. The tumour, which was easily removed, proved to be a fibroma. Uninterrupted healing took place.

On the evening of the same day there was some increase in power in the right hand. Four days later there was distinct improvement in the power of both hands and the right leg, and the anæsthesia was less marked.

Six weeks after the operation the patient was able to walk with support; the right leg was ataxic.

When seen eight months after the operation he could walk well with a stick, could jump off either leg, and his hands had almost completely regained their former strength; he still, however, complained of occasional numbness in the parts which were previously most anæsthetic.

The diagnosis is considered in great detail. A number of charts are given illustrating the distribution of the anæsthesia, also a sketch showing the position of the tumour *in situ* as seen at the operation.

EDWIN BRAMWELL.

**THE RESULTS OF THE OPERATIVE TREATMENT OF SPINAL  
(70) TUMOURS.** A Critical Review. R. T. WILLIAMSON, M.D.,  
F.R.C.P.

AFTER briefly reviewing the statistics of Sir William Gowers and Sir Victor Horsley, of Allen Starr and of Schlesinger, the writer has collected and tabulated 53 cases of spinal tumour upon which operations for removal were performed. These cases have been collected from the medical literature subsequent to 1887. Short details are given of 27 of these cases in which partial or complete recovery followed operation. An instance of successful removal of extra-dural hydatid cysts in a child aged two and a half years which occurred under the writer's care is also reported.

The remarkable rarity of syphilitic tumours of the spinal cord is emphasised.

The brilliant results that are often attained by early surgical interference where the growth occurs in connection with the meninges are pointed out, the importance of early and correct local diagnosis and exploration in all cases suggesting pressure upon the spinal cord are insisted upon.

The highest limit of the slightest sensory change should be taken as indicating the segmental level of the lesion. Frequently the lesion is found one or two segments higher than that indicated by detectable anæsthesia.

J. S. COLLIER.

**ACUTE POLIOMYELITIS AND ENCEPHALITIS.** By FREDERICK  
(71) E. BATTEN, *Lancet*, Dec. 20, 1902, p. 1678.

UNDER this title the author describes a group of cases which he considers should be classed together, although they may present clinical features which vary according to the part of the nervous system affected, yet should be regarded as one disease since they are due to the same morbid and pathological process. He divides these cases into three main groups—(1) acute polio-encephalitis superior; (2) acute polio-encephalitis inferior; and (3) acute polio-myelitis anterior—but he is careful to point out that although it is convenient to describe such groups, yet there are numerous cases which exhibit symptoms characteristic of two or even all of these groups. Under the first heading, polio-

encephalitis superior, he describes cases in which the frontal, motor and occipital cortex are affected, and also cases in which the lesion probably exists in the cerebellum. Under the second heading, polio-encephalitis inferior, he describes cases in which the various nuclei of the cranial nerves are affected; and under the third heading, polio-myelitis anterior, cases in which the grey matter of the anterior process below the medulla is affected. The various groups are illustrated by cases, some of which have been verified by post-mortem and microscopical investigation—others are based only on clinical evidence.

He raises the question whether anterior polio-myelitis ever occurs in intrauterine life, and quotes a case which on clinical grounds he regards as such, but he is unable to bring forward any pathological evidence on the subject. He states that the morbid anatomy of the disease is characterised by engorgement and thrombosis of small vessels, perivascular exudation, minute extravasation of blood and much round-cell infiltration of the neighbouring tissues.

With regard to the pathology of the disease, he discusses the question whether the condition is due to a definite specific infection producing an acute inflammation or is due to a vascular thrombosis dependent on some altered blood condition, such altered blood condition being due to various causes and not dependent on one specific infection. He concludes that the condition is due to a thrombosis occurring in the finer arteries, and that such thrombosis is dependent on an altered blood condition.

EDWIN BRAMWELL.

**ZUR SYMPTOMATOLOGIE UND DIAGNOSTIK DER GESCH-  
(72) WÜLSTE DES STIRNHIRNS.** Von EDUARD MÜLLER,  
*Deutsche Zeitschrift für Nervenheilkunde*, Bd. 22, 5 u. 6 Heft.  
S. 375.

THIS interesting paper consists of a careful analysis of 164 cases of frontal tumour collected, with one exception, from the literature of the subject. An inquiry of this nature yields necessarily a very large number of statistical facts from which many deductions can be made, but it is impossible here to quote more than a few of the most important.

Dr Müller finds that in a few cases of frontal tumour there is early general wasting of the body in spite of the absence of vomiting and the ingestion of plenty of food, an observation which is of interest when taken in conjunction with the experiments of Modica and Andenio on the relation of the frontal convolutions to met-

abolism. An analogous condition is present in some forms of mental disease. A consideration of the disturbances of body temperature gives no support to Schüller's theory concerning the existence of a thermic cortical centre in the second frontal convolution.

Apart from such obvious conditions as local œdema and thinning of bone the examination of the cranium is not very helpful, but percussion gives useful results in a few instances.

Epilepsy occurs in one-third of the cases of frontal tumour, but the site of the neoplasm does not seem to influence its occurrence except when the convulsions are Jacksonian in type. In six patients Jacksonian attacks occurred in which the conjugate deviation of the eyes and head was the first movement, and a careful consideration of these cases leads the author to conclude that: (1) the centres for conjugate deviation of the eyes and for lateral turning of the head lie in the hinder part of the second frontal convolution; (2) the head-turning centre lies just below the eye-turning centre; (3) in Jacksonian attacks the nervous discharge spreads from the head and eye centre to those representing the movements of the face and arm. Most worthy of note is the fact that spasm of the neck and trunk muscles occurs only in cases of tumour of the right or of both frontal lobes; this deduction is made from ten cases which came to autopsy.

General weakness occurs early and is explained by the author on the ground of the proximity of the motor region of the brain. Headache is wanting in only six per cent. of the cases; it is most frequent in the forehead, and in twenty instances more pronounced on the side of the tumour. Occipital headache and stiffness of the neck is very infrequent. Optic neuritis is found in nearly 80 per cent. of the patients; in five it was unilateral, and in four of these it corresponded to the side of the lesion in the brain. Exophthalmos occurs in four cases and ocular palsies in 15 per cent.

E. FARQUHAR BUZZARD.

#### **KLINISCHER UND ANATOMISCHER BEITRAG ZUR ERKRANK-**

(73) **UNG DER NEUROGLIA.** M. ROSENFELD, *Monatsschrift für Psychiatrie und Neurologie*, Oct. 1902.

THE author deals here with four cases of glioma of the brain with details of the subsequent examination, and he also gives the clinical histories of two other cases of cerebral tumour. He mentions that it is sometimes difficult to distinguish between a diffuse sclerosis and a glioma, and quotes Ziegler, who says that there is

no sharp line to be drawn between diffuse sclerosis, glioma, and neuroglioma, and also that intermediate forms between the different pathological conditions of the neuroglia are met with. The clinical history of his first case showed that the patient had suffered from epilepsy, paresis of the right limbs, increased reflexes, optic neuritis, disturbed sensation, and motor aphasia, and had exhibited marked mental deterioration. Post-mortem a growth in the left central region was found which implicated the cortex. His second case suffered from epilepsy and complete amaurosis from optic atrophy. Here there was no mental disturbance. Post-mortem a growth was seen in each frontal lobe. In his third case there was found post-mortem a growth in the left frontal and central regions which had caused epilepsy and paresis of the right side. There was marked mental change. The fourth patient was congenitally weak-minded, and had suffered from epilepsy from his childhood. He had several long series of fits, and eventually died in the condition of status epilepticus.

The growths consisted of glia fibres and numerous vessels; nerve elements were scattered throughout the tumour. The glia fibres varied in thickness and in shape; they were arranged generally in an irregular network, but near the vessels they were more or less collected into bundles. Sometimes they lay across or encircled the vessels, but they had no definite connection with them. The glia cells were scarce generally, but around the vessels they were in some places collected into groups, "Strahlenkronen." They showed a normal shape, size and staining reaction. No astrocytes or monster glia cells (Storch) were seen. The vessels were very numerous; they showed no regressive changes.

In some cases, and especially in Case iii., in which the tumour was a cell-rich, soft, vascular glioma, small hæmorrhages were found.

The nerve elements were generally normal in appearance, and were simply separated into groups by the fibres forming the tumour.

In some places secondary degenerations were seen. The author drew attention to two points in connection with these cases: first, that regressive changes in the tissues of which the tumour was composed were absent or very slight; second, that all the cases suffered from epileptic attacks, and Case iv. died in the condition of status epilepticus. In Case i. the epileptic attacks had a cortical origin; in Case ii. they were due to pressure; in Case iii. the convulsions were general and were present throughout the illness; and in Case iv. they occurred in series.

Many observers deny that gliomata possess an irritative action and give rise to epilepsy; they say that apoplectiform attacks are

much more characteristic, and that focal symptoms frequently appear simultaneously with the apoplectiform attack.

In Cases i. and iii. there was marked mental deterioration ; in Case ii. there was none ; Case iv. was congenitally weak-minded.

R. G. Rows.

**THE SURGERY OF BRAIN TUMOURS FROM THE POINT OF  
(74) VIEW OF THE NEUROLOGIST, WITH NOTES OF A  
RECENT CASE.** By CHARLES K. MILLS, *Philadelphia Med.  
Journ.* Nov. 29, 1902.

THE author has seen twenty-two cases in which operation was performed after the diagnosis of tumour or cyst. "I do not make the slightest claim to the surgeon's prerogative, but the frequent onlooker, especially if he is personally interested, may acquire the right to discuss matters critically." Dr Mills believes that the percentage of successes will be much increased with more exact localisation, more precise cranio-cerebral topographical methods, and more perfect surgical technique. A careful study of the order of development of the symptoms is of great importance for purposes of localisation in exactly fixing the limits of an operation.

The author prefers the Anderson-Makins method of locating the chief cranio-cerebral landmarks. In a case of operation his procedure is, whenever possible, not only to locate the tumour, but also to fix the limits and direction of the osteo-plastic flap. He is strongly in favour of the osteo-plastic operation, and the use of the Stellwag trephine. Experiments on the cadaver, "which will probably be described by Dr Keen," are referred to, to show the possibility of operation for tumours of the pons, crus and cerebellum. From these experiments it was evident that these parts could all be thoroughly exposed.

The history of a case of tumour of the Rolandic area is detailed. In this case an irregular shadow was apparent on X-ray examination in the position of the growth. Large doses of potassium iodide and mercury having proved ineffectual, the patient was operated on and the tumour successfully removed by Dr W. J. Hearn. It proved on microscopical examination to be a gumma. The patient made a perfect surgical recovery, and four weeks after the operation his headache, and the Jacksonian attacks from which he formerly suffered had quite disappeared, his eyes had improved, and he was in all respects in excellent condition.

EDWIN BRAMWELL.

**CONTRIBUTO ALLO STUDIO DELLE IPOTERMIE NEGLI  
(75) EPILLETICI. C. BESTA, *Riv. Sperm. di Fren.*, fasc. 4, 1902.**

THE patient on whose history Besta's remarks are based was a man of twenty-eight years of age who had suffered from epilepsy for ten years. At first the fits were nocturnal followed by profound sleep and on wakening by much mental confusion. After two years they occurred also by day and were often followed by delirium in which the patient was very dangerous.

Later his intelligence became reduced and at the time his abnormality of temperature attracted attention he was silent and taciturn.

On the 21st January his temperature became subnormal and remained so until February 6, when it rose again to 37°. During these eighteen days there were irregular oscillations between 35.1° and 36.4° except on February 1, when the temperature reached 36.9°. On the 26th February he had three fits, when the temperature reached 37.5° but dropped again. He had one other fit on January 27th without rise of temperature.

During this period of time he did not suffer from vertigo nor from any other special symptom: his general condition was good, and his mental state remained unvaried.

Besta believes that the lowering of temperature is due to some special condition and draws attention to Ceni's statement that epileptic serum can cause a lowering of temperature. The fact that the patient's temperature remained subnormal even after three fits, according to Besta supports Ceni's view that there exists no relationship between the drop of temperature and the fits. Regarding the question as to whether the fits are due to progressive accumulation of toxins, the author points out how if that were so the temperature ought to go down progressively and then rise again after the fits, if by them the toxins in the circulation were reduced. But he points out how the subnormal temperature persisted even for ten days after the fits without any new ones, and that therefore the toxins must have remained in the circulation. He then points out that his case shows how the psychomotor centres react at once, and the vasomotor centres slowly and independently of each other to the same agent, a fact insisted on by Ceni, who has further demonstrated that the serum taken during a subnormal crisis has not any greater power of reducing the temperature than that taken during normal temperature.

DAVID ORR.

**SULLA DISVULNERABILITÀ DEGLI EPILETTICI. V. TIRELLI,**  
(76) *La Riforma Medica*, 1902, vol. iv. No. 50.

TIRELLI remarks at the beginning of his paper on the extraordinary power of recovery which epileptics possess from severe injuries, even where there has been every chance of septic infection, and has undertaken the following research in order to see if there exist in the serum of this class of patient special conditions which hinder the development of pyogenic germs.

He obtained serum from epileptics, and also from patients not epileptic, and to each he added a virulent culture of a staphylococcus. The former he called B, the latter A. The cultures grew in the B mixture more slowly than in A.

A series of experiments was conducted upon animals, with the intention of determining if the epileptic serum exerted any influence upon the effects of inoculation.

The author found that culture A killed the animal into which it was injected in from 36-60 hours, while the staphylococcus mixed with epileptic serum took from 83-142 hours to produce a fatal result, thus showing a considerable protraction of the lethal period. Further, in two experiments he injected sterilised epileptic serum at various times after the injection of solution B, and found always a slight improvement in the symptoms, and that the death of the animal was delayed.

Tirelli then treated an animal with successive injections of epileptic serum, to see if it acquired an immunity to the action of the staphylococcus, and found that the animal so treated withstood the infection for double the time of one untreated thus previously.

He concludes from his work that: (1) epileptic serum has a superior bactericidal power to ordinary serum, and can attenuate the development and virulence of the staphylococcus pyogenes aureus; (2) this antipyogenic property explains the resistance of epileptics to severe injuries; and (3) probably epileptic serum possesses a curative and immunising property against staphylococcic infection.

DAVID ORR.

**RECHERCHE D'UN TRAITEMENT DE L'EPILEPSIE ESSEN-**  
(77) **TIELLE** Par le Dr LÉON CERF, *L'Anjou Médical*, vol. ix. July 1902.

In the treatment of idiopathic epilepsy Dr Cerf has obtained such striking results from the use of thyroid extract, that he has been led to suggest the possibility of an epilepsy of thyroid dystrophy.



The good results of thyroid treatment which he now records are not, however, sufficiently complete to enable him to formulate this new theory without reserve.

He describes the case of a child aged five and a half years in whom the first fit occurred at five months.

Fits occurred every day at first; then there were occasional intervals lasting eight days at the most, after which the attacks were more numerous and violent—five or more fits daily.

When first seen the child showed some signs of backwardness; he slavered continually, was extremely dirty in his habits, and was only able to utter inarticulate sounds.

Thyroid treatment was commenced with 20 centigrammes of Baumann's iodothyrene daily, in divided doses, and was continued without interruption for ten consecutive months.

Shortly after the commencement of the treatment, the fits diminished both in number and severity, and in a month ceased altogether. For eighteen months afterwards, up to the time of Cerf's last visit, there had not been a single fit. The child grew and developed normally and advanced intellectually.

Here is an instance of the disappearance of "epileptiform" fits in a child under the influence of thyroid treatment.

As Cerf puts it, "evidently what had been wanting for more than five years had in ten months been brought forward, and we are bound to attribute the credit, in part at least, to the thyroid treatment."

After this experience, four cases of epilepsy were treated with thyroid, and in all it appeared to have an undeniable influence on the fits. Cerf suggests that it would be well to try thyroid extract and the fresh gland, as well as Baumann's iodothyrene.

H. O. NICHOLSON.

**BEITRAG ZUR KENNTNISS DER MYASTHENIA GRAVIS MIT  
(78) BEFUND VON ZELLHERDEN IN ZAHLREICHEN MUS-  
KELN.** Von Dr RICHARD LINK, *D. Ztschrift. f. Nervenheilkunde*,  
Bd. 23, H. 1 und 2, 1902, S. 114.

AFTER referring to the two important autopsies recently recorded by Laquer and Weigert, and by Goldflam, the author announces that he has met with a case in which similar changes in the muscles were found post-mortem. The clinical history of the case is recorded in full. The patient, a man aged 43, presented the following characteristic symptoms of myasthenia gravis: a varying degree of ptosis; ocular pareses and diplopia; a mask-like face; dysphagia; severe attacks of choking; a condition of wide-spread

paresis and abnormal muscular fatigability. The myasthenic reaction was well marked. There was no muscular atrophy, and no objective sensory disturbance, nor was there evidence of disease in any of the other organs. The patient died of respiratory failure four months after the appearance of the first symptom.

At the autopsy the central nervous system appeared normal to the naked eye, excepting that the pia mater was somewhat injected. A persistent thymus 3 cm. in length was found. There was some bronchitis, and there were some patches of commencing bronchopneumonia.

A careful microscopic examination of the nervous system was made by the methods of Weigert-Pal and van Giesson. The following regions were examined: motor cortex from the arm centre; sections through the aqueduct of Sylvius in the region of the ocular nuclei; sections through the anterior part of the floor of the 4th ventricle; pieces of the cervical and lumbar regions of the spinal cord; both musculo-spiral and sciatic nerves, and the right median nerve. Excepting that there appeared to be an abnormal number of cells in the neighbourhood of the central canal of the spinal cord, nothing unusual was met with.

The thymus gland was perfectly normal in structure; in particular there was no evidence of malignant disease, and its blood-vessels showed no pathological changes.

In both internal recti muscles, the right external rectus, both long supinators, both deltoids and the right tibialis anticus, all of which appeared quite healthy to the naked eye, groups of small round or oval, uninucleated cells with little protoplasm were found on microscopical examination. These were situated in part within the perimesium, in part between the muscle fibres. In the neighbourhood of these cell groups, and sometimes within them, were small blood-vessels; in one preparation a recent hæmorrhage was noted. The transverse striation of the muscle was everywhere well preserved, and only here and there muscle fibres lying within the groups were somewhat shrunken. No organisms were detected with bacterial stains, nor could eosinophile granules be seen in sections stained by Leishman's method.

In a number of other muscles, which were also examined microscopically, including the diaphragm and a papillary muscle from the heart, these cell groups were not found.

As to their nature, the author opposes the view advanced by Weigert and supported by Goldflam that they are metastases, for in his case there was nowhere evidence of a malignant tumour. He considers that they are not inflammatory because of the absence of myositis, the muscle fibres in the immediate neighbourhood of the cell groups were quite normal, and since no bacteria were detected in them.

Weigert has suggested a possible relation between the thymus gland and myasthenia gravis. The author refers to two experiments which he has made in relation to this point. (1) An extract of fresh thymus was injected into a rabbit with a negative result. (2) He grafted a thymus from one rabbit under the skin of another animal of the same species. The animal was killed twenty-seven days later and the muscles examined microscopically. They were found to be perfectly normal.

Link suggests that possibly these cell groups may interfere with the lymph flow and in this way produce the myasthenic symptoms. In favour of this theory he points out that if the upper arm is bandaged with an elastic bandage, care being taken not to obliterate the radial pulse, the muscles of the forearm on that side when exercised become much more speedily fatigued than those of the opposite arm.

Two figures are included in the text to illustrate the changes described in the muscles.

EDWIN BRAMWELL.

**UEBER AGRAMMATISMUS ALS FOLGE VON HERDER-**

(79) **KRANKUNG.** Von A. PICK, *Ztschr. f. Heilk.*, Bd. 23, H. 2, 1902.

THIS case is related because it confirms the author's idea that agrammatism is due to a localised lesion in the temporal lobe. The symptoms exhibited were those of that form of agrammatism which is characterised by the exhibition of the telegraphic style of speech and writing, and several specimens of this are given. The examination of the brain revealed distinct atrophy of Broca's convolution and the anterior part of the left superior temporal convolution. The author considers that the latter was precedent in point of time, and gives reasons for this conclusion. A plate shows the condition of the two cerebral hemispheres.

J. MIDDLEMASS.

**EIN FALL VON AKUTER EXTERIORER OCULOMOTORIUS-**

(80) **LÄHMUNG AUF NEURITISCHE BASIS.** Von L. v. FRANKE HOCHWART, *Arb. aus dem Neurolog. Institute an der Wiener Universität*, herausgegeben von Prof. Heinrich Obersteiner, H. ix. 1902, S. 322.

THE patient was a man, sixty-seven years of age, who, with the exception of an attack of malaria in youth, had enjoyed excellent health till five years ago. Without any previous symptom, he

awoke one morning to find his right side paralysed. The face was not involved, though speech was impaired for five days. After nine months he could write again, and do his work, though weakness remained in the right leg. A year ago, while writing, patient suddenly became aware of a dimness in the right eye. This was followed in half an hour by diplopia. In the evening he could not open the eye. On elevating the lid with the finger, he found that the diplopia was persisting. There was no headache, giddiness, or vomiting, and no involvement of speech. Syphilis was absolutely denied.

On examination it was found that the right lid could with difficulty be lifted about 7 or 8 mm. Closure was normal. The eye was deviated slightly outwards, and could not be turned inwards beyond the middle position. The pupil reacted to light and for accommodation, and the fundus was normal. The patient died suddenly a few days later.

P.M.—A high degree of atheroma and calcification was found in the coronary arteries. The heart muscle showed fatty degeneration. There were no signs of syphilis in any organ.

The hemiplegia was accounted for by an old thrombus in the artery of the Sylvian fissure, which, on microscopical examination, showed a peculiar cartilaginous formation (*cf. Marburg, Centralbl. f. allgem. Path. u. path. Anat.*, Bd. xiii. S. 300).

As far as the right oculomotor nerve was concerned, careful microscopical scrutiny of the cells in the nucleus revealed no pathological change; nor was anything found in the ciliary ganglion on either side. The trunks of the two third nerves were examined. The left was normal. The right showed some disintegration of nerve fibres, with great numbers of round cells between the bundles. The vessels in the connective tissue of the sheath were engorged with blood. Hence the diagnosis of acute interstitial neuritis of the third cranial nerve on the right side.

The reporter of the case excludes, as a cause, syphilis, inflammation of the neighbouring membranes, compression from a dilated artery, and is inclined to consider the case as one of *neuritis multiplex arteriosclerotica*, in which, however, only one nerve has been involved.

S. A. K. WILSON.

**OPHTHALMOPLÉGIE CONGÉNITALE.** ALPHONSE PÉCHIN, *Revue* (81) *Générale d'Ophthalmologie*, Nov. 1902.

CASES of congenital ophthalmoplegia are certainly extremely rare, though ptosis is far from infrequent, and along with it defect of upward movement is often enough to be seen. The case recorded

by Péchin was that of a youth of 20, who had complained of difficulty in moving his eyes ever since the age of 15, when he first began to earn his living. It was probable from the history, though not, it must be admitted, absolutely certain, that the defect had existed long before that time, and there had been no illness during childhood which could have given rise to the condition. The utmost range of movement of which the eyes were capable was only about one millimetre: they were practically immovable either in a vertical or horizontal direction, and appeared as though modelled in wax. The lids hung down so far that in order to see any object straight upon the level of the eyes he had to tilt back the head. By exertion of the occipito-frontalis he was able to raise them a little, but the effort was feeble and curiously evanescent: it could not be kept up for more than five or six seconds. The facial muscles were all in a similar state of feebleness, but the pupils acted normally, accommodation was quite active, vision on correction of his myopia was up to the normal, and the fields of vision were unrestricted. There was no indication of hereditary syphilis.

W. G. SYM.

(82) **PAIN.** JAMES MACKENZIE, *Brain*, Autumn 1902, p. 368.

THE purpose of the paper was to give the reasons in justification of the following definition:—*Pain is a disagreeable sensation due to stimulation of some portion of the cerebro-spinal nervous system, and referred to the peripheral distribution, in the external body wall, of cerebro-spinal sensory nerves.*

This definition implies that the sensation of pain, from whatever source the stimulation arises, whether in the brain, in the spinal cord, in the posterior root ganglia of spinal nerves, in the viscera, or in the external body wall, is invariably referred to the peripheral distribution of a sensory cerebro-spinal nerve, and that, therefore, pain is a phenomenon peculiar to the cerebro-spinal nervous system.

In the above definition the division of the nervous system into a cerebro-spinal and sympathetic is maintained, as the clinical facts point to the conclusion that the sensation of pain (and probably other sensations) does not arise from stimulation of the sympathetic nerves, unless through the intermediary of the cerebro-spinal nerves. In developing the argument in favour of this definition, reference is made exclusively to the pain and associated symptoms arising from stimulation of the viscera.

Experimental observations on animals are practically of no avail in determining this question. Such experiments as have shown the various organs to have been insensitive, prove only that the stimulus employed had not been adequate to produce pain. On

the other hand in such experiments as have demonstrated that the stimulus has been adequate, and where the animal has shown symptoms of pain, no evidence is available to determine in what region the pain was felt.

Trustworthy evidence is afforded only by the study of the sensations felt by the conscious human subject.

In the clinical examination a universal source of error has arisen through the assumption that any given organ was sensitive because pain was felt when pressure was applied over the organ. The fact that the tissues forming the external body wall were being stimulated at the same time has been overlooked.

The pain arising from pressure over an organ is due to hyperæsthesia of the skin, muscles or other structures in the external body wall. Cutaneous hyperæsthesia occurs in two forms, a superficial elicited by gently stroking the skin with the head of a pin, and a deep elicited by gently pinching the skin. The deep kind of hyperæsthesia may be present without the superficial. The areas of hyperæsthesia occur in the distribution of those nerves whose spinal centres are in communication with the sympathetic nerves supplying the affected viscus. Visceral disease may cause contraction of muscles in the external body wall—the *viscero-muscular reflex*. The muscles of the abdominal wall may contract universally as in general peritonitis, or in small sections. In the latter case it is difficult to distinguish between the hardened muscle and a tumour in the abdominal cavity.

The effect of the direct application of stimuli to the viscera shows that exposed organs in the conscious subject are insensitive to most stimuli; but when an adequate stimulus is applied, the resultant pain is invariably a referred pain.

The suggested explanation of the manner in which pain and other symptoms arise in visceral disease is as follows:—

In the performance of the functions of an organ a stream of energy is continuously passing from the organ by the sympathetic nerves to the spinal cord. The subject is unconscious of any sensation during the routine performance of these functions. When from any cause an excessive stimulus is applied to these nerves, they convey an excess of energy to the centre of other nerves in the spinal cord or elsewhere. These centres being thus stimulated give forth results according to their functions. Thus a sensory nerve will give rise to pain felt in its peripheral distribution; a muscular nerve will give rise to muscular contraction, as in the hardening of the abdominal muscles in peritonitis; a secretory nerve will stimulate its organ to excessive action, as when an abundant flow of saliva occurs during an attack of angina pectoris, etc.

AUTHOR'S ABSTRACT.

**DAS VERHALTEN EINIGER REFLEXE BEI GESUNDEN UND  
(83) BEI TABES. BEMERKUNGEN ZUR FRÜHDIAGNOSE  
DER TABES.** Von Dr JENO KOLLARITS, *D. Ztschrift. f.  
Nervenheilkunde*, Bd. 23, H. 1 und 2, 1902, S. 89.

In this contribution the author records his observations on the constancy of some "reflexes" in healthy individuals and in tabetics. He has examined the tendo-Achillis jerk, the knee-jerk, the triceps-jerk and the scapulo-humeral reflex in 1000 individuals, none of whom were suffering from any form of nervous disease. In every instance he was able to obtain all four "reflexes," "so that the absence of these reflexes can always with certainty be regarded as pathological."

In examining the scapulo-humeral reflex he points out that although in the erect position it is sometimes impossible to obtain it, if the patient bends forwards his hands hanging loosely by his sides, it can always be elicited in health. In testing the Achillis-jerk he makes the patient kneel with the leg to be tested on a chair, the other foot rests on the ground and maintains the weight of the body; in this way he finds that the muscles are usually more perfectly relaxed than by any other means. Before testing the triceps jerk, the tendon of the muscle should first be felt for. The observer directs his attention not only to the movement of the arm but also to the triceps muscle, for a contraction frequently occurs in the long head of the triceps without producing any movement of the forearm. According to the author, want of attention to this point accounts for the discrepancy of opinion which exists as to the constancy of this jerk in health.

Reinforcement is just as important in the case of the jerks of the upper as in those of the lower extremities. In the former case, pressing the knees together or grasping firmly with the opposite hand will be found a satisfactory means.

The author has also examined the above-mentioned reflexes in 100 cases of tabes. His results are as follows:—

Achillis-jerk.	Both absent in 65;	one absent in 5 ;	both present in 30
Knee-jerk.	" 56	" 4	" 40
Triceps-jerk.	" 43	" 10	" 47
Scapulo-humeral reflex.	35	" 2	" 63

A table is appended to the paper in which details of these cases of tabes are given.

EDWIN BRAMWELL.

**REFLEXES: THEIR RELATION TO DIAGNOSIS IN RHEUMATOID ARTHRITIS.** By R. LLEWELYN JONES, *Lancet*, Dec. 1902.

CASES in which the disease is asymmetrical are the most suitable for the study of the reflexes. As a general rule the deep reflexes are exaggerated on the affected side; although in some very few cases a total loss of reflex action, with muscular reaction of degeneration, has been observed. The behaviour of the superficial reflexes is more uncertain. In the upper limb it is observed that disease of particular joints is accompanied by exaggeration of particular reflexes. For example, if the ring and middle fingers are affected the flexor tendons at the wrist give a brisker response than the extensor. If the index and thumb are affected the reverse is found. In the lower limb the knee-jerk is always exaggerated, no matter what joints are diseased, and this exaggeration may even amount to a true clonus of the quadriceps. With regard to the plantar reflex, when the knee-joint is involved a marked exaggeration is always present. If the ankle joint be affected, however, either alone or in addition to the knee, the plantar reflex is usually sluggish; this being probably explained by the cold, moist condition of the sole of the foot. A definite extensor plantar response has been obtained in some cases. A curious agreement is observed between the plantar and gluteal responses even in cases in which the ankle is alone affected, and wasting of the gluteal muscles is frequently found on the affected side. Precisely similar changes are found in the scapular reflex and shoulder muscles when the wrist joint is diseased. When the plantar reflex is sluggish hyperalgesic areas are found on the sole, the buttock, and the outer part of the thigh of the affected side. Ankle clonus may be present, usually only in the more acute forms of the disease. The abdominal reflexes are found to be exaggerated on the side corresponding to the diseased joints. Somewhat indefinite disturbances have been noted in connection with the organic reflexes. Myotatic irritability and vague nervous symptoms, such as tinglings, cramps and local syncope, frequently precede the actual onset of arthrodial trouble, and these persist for some time after the disappearance of joint mischief in those few cases which recover. Again, the continuance of reflex exaggeration in cases which appear to be quiescent is always of bad omen.

On consideration of the phenomena observed in muscles, reflex action and skin when any particular joint is affected it would appear that the parts affected together are usually under the control of one segment of the spinal cord. This suggests the possibility of the disease being of the nature of a toxæmia of the central nervous system.

HENRY J. DUNBAR.



**ZUR PATHOLOGIE DER HAUTREFLEXE AN DEN UNTEREN**

(85) **EXTREMITÄTEN.** H. OPPENHEIM, *Monatsschrift für Psychiatrie und Neurologie*, Bd. xii. 1902, pp. 421 and 518,

If the inner side of the leg be stroked firmly from above downwards with the handle of a percussion hammer, along the posterior border of the tibia or a little posterior to it, commencing about a hand's breadth below the knee and going downwards nearly to the ankle, the result in healthy individuals and in cases of functional nervous disease is either negative or it produces a plantar flexion of the toes. Sometimes all the toes become flexed, sometimes only the outer ones. The patient's attention must meanwhile be diverted, so as to eliminate any voluntary movement.

But in lesions of the pyramidal tract, as in spastic paraparesis or hemiparesis, the reflex movement produced by the above method of stimulation consists in extension of the toes and abduction or adduction of the ankle. In such cases the extensor longus hallucis is generally the first muscle to respond, next the tibialis anticus and extensor communis digitorum, and, lastly, in a small proportion of cases, the peronei. Sometimes one muscle, sometimes another, predominates in activity, so that the resulting movement varies in different cases, but in pyramidal lesions the response is never of the normal flexor type. Up to a certain degree, the more rigid the muscles, the better marked is the phenomenon, but if rigidity be excessive it may totally prevent the reflex. The duration of the reflex is not always a momentary one, occasionally it is as much as thirty seconds.

Oppenheim is of opinion that this reflex is not identical with Babinski's toe-phenomenon, inasmuch as it is often present in cases of spastic paresis where Babinski's reflex is doubtful; moreover it does not occur in the small proportion of healthy persons who exhibit the Babinski reflex; finally the involvement of the tibialis anticus and occasionally of the peronei is another point of difference from the phenomenon of Babinski. Its diagnostic significance is equal to that of the "phénomène des orteils."

Sometimes when stroking the leg fails to produce Oppenheim's reflex, it may be elicited by pinching up a fold of skin in the same region.

PURVES STEWART.

**PSYCHIATRY.**

**UEBER DIE ACUT VERLAUFENDEN ERKRANKUNGEN AN**  
(86) **DEMENTIA PARALYTICA.** Von Prof. Dr BUCHHOLZ, *Arch.*  
*f. Psychiat. u. Nervenkr.*, Bd. xxxvi. H. 2, S. 427, 1902.

IN estimating the relative frequency of cases of acute general paralysis, care must be taken to exclude all those of short duration in which death was due to some cause other than the disease itself or a complication directly arising from it. Moreover the term is used by different authors to denote various classes of cases, whereas those which should be included under it are cases of acute paralysis commencing in persons previously healthy (although it is seldom possible to be sure that there have been absolutely no preceding symptoms). The objects of the present research were to determine the frequency of these cases and to test the truth of the statement made by various authors that they may be divided into two classes, viz., those in which there is merely an abbreviation of the ordinary course of the disease, and those in which the disease takes a special colouring, coming somewhat to resemble acute delirium.

Out of 326 male general paralytics admitted to the Marburg Asylum between 1876 and 1900, only 24, or 7·36 per cent., died within the first year. Of these, however, 9 died of intercurrent diseases, leaving only 15 which could be included in this category as defined above. Nine of these 15 cases ran a course which differed from that of ordinary general paralysis only in its brevity; in 2 other cases the disease ran its ordinary course for a time and then suddenly ended in a stage of the severest excitement; while in the remaining 4 the symptoms throughout, or after a short prodromal stage, resembled those of acute delirium, though some of the clinical phenomena, as well as the post-mortem appearances (which in two of the cases are described with great minuteness), showed the true nature of the disease. Two other cases are shortly given, but are not included in the above numbers, as their nature is very doubtful.

Out of 50 female cases admitted during the same period, 6 died in the first year of the attack, but only one of these from the disease itself, though a second, who died of suffocation, was probably a case belonging to the acute category. (These two cases have already been described by Tuczec.) Thus the proportion of female general paralytics dying in the first year is larger than that of males, being 12 per cent.; but the numbers of the former are too small to possess much significance.

It would perhaps be well to classify the first 11 cases under

the name of *acute paralysis of rapid course*. In the last two of these cases the excitement developed, in one after a course of mercurial treatment; in the second after an apoplectiform seizure together with a severe phlegmon. There thus remain only 4 cases of true galloping or fulminant general paralysis amongst the males, and 1 female case, so that this particular form belongs to the rarities.

As regards the pathology of the 4 male cases, the appearances corresponded with the clinical symptoms. No very thorough examination was made of the first or second, but the congested state of the cerebral vessels argued for an acute process in both, while the presence of sclerosis in the cord of the second case showed that this was combined with a more chronic degeneration. The third case also showed congestion, and a widespread pachymeningitis also indicated the acuteness of the attack. The cord showed two distinct sets of changes; a fairly extensive but incomplete degeneration of the posterior roots and columns, and also of Clarke's columns, etc., extending to the lateral columns, the whole being of a subacute character, as was degeneration of the cells of the anterior horn; and a very acute process of a myelitic nature, marked by swelling of the axis-cylinders. A cellular infiltration of the soft membranes of the cord was found both in this and the fourth case. The latter showed a degeneration resembling in all essentials the sclerosis of tabes, and obviously chronic; and side by side with this a recent florid process, differing from the former in its acuteness, and from myelitis in the absence of small round cells (as the granule cells present were, in the author's opinion, modified neuroglia cells which had taken on phagocytic functions). Probably, however, this process would also end in sclerosis. The appearances showed that the chronic disease in this case received an impetus and became acute, and this impetus was probably given by a course of mercurial inunction, as in one of the cases previously mentioned. (Similar results from this treatment have been recorded by Zacher, and it must consequently be regarded as dangerous.) From the pathological appearances, this fourth case is therefore probably not a true example of the fulminant variety.

Notwithstanding the differences, this rapid form of disease is genuine general paralysis. It may be divided into *acute general paralysis* and *galloping or fulminant general paralysis*, the former being marked by an abbreviated and stormy course, while in the latter a short prodromal stage is followed by symptoms resembling those of acute delirium. In addition to these, however, there is a class of cases in which after a more chronic course the delirious symptoms set in and seal the fate of the patient.

W. R. DAWSON.

**ON THE TYPHOID PSYCHOSES.** By CLARENCE B. FARRAR, *Amer. (87) Journ. of Insan.*, July 1902, p. 17.

THE author reports four cases of insanity associated with typhoid fever which have come under his observation at the Sheppard and Enoch Pratt Hospital at Baltimore, and he gives an account of the Typhoid Psychoses in general, which, following Kraepelin, he arranges in three main groups:—

(1) *Initial Delirium*.—The rarest, most rapid, and worst form, over 50 per cent. ending fatally. It is especially apt to occur in patients with a psychopathic heredity; and it is essentially a severe intoxication, as evidenced by the Nissl findings in the large pyramidal cells. Alzheimer and Sander, however, have described similar cell changes in cases of typhoid fever and pneumonia without insanity. The diagnosis is often difficult as there may be merely delirium, with no fever for several days, these being very grave cases; in equally fatal cases there may be delirium with high temperature, these exhibiting on post-mortem examination an acute hæmorrhagic meningo-encephalitis.

(2) *Febrile Psychosis*.—The commonest and most hopeful form. It arises during the periods of invasion, advance, or resolution of the fever, and more or less comes and goes with the pyrexia, although 25 per cent. of cases persist for a longer or shorter time after the fever is over. The special pathogenetic factors of this form are, in addition to heredity and toxæmia, especially the pyrexia and possibly cerebral hyperæmia or anæmia or œdema.

(3) *Asthenic Psychosis* arises during convalescence, is especially seen in long protracted cases, developing upon a special basis of exhaustion, anæmia and malnutrition, and is of doubtful prognosis. Lastly, after an attack of typhoid fever there may remain for months or years a condition of irritable weakness of the nervous system, upon which may develop a late psychosis, also of doubtful prognosis.

Conversely, it has been observed that an attack of enterica during the course of a psychosis or a neurosis may be followed by improvement or even recovery of the latter, probably owing to the metabolic revolution produced by the fever.

The author analyses the symptoms of typhoid insanity, and concludes that it presents no distinctive clinical picture; "the elements of intoxication, infection, temperature, exhaustion, anæmia, of whatever origin, may produce indistinguishable appearances."

C. C. EASTERBROOK.

**DIE PSYCHOSEN DER LANDSTREICHER.** WILLMANS, *Versamml.* (88) *d. S. W. deutsch. Irrenärzte.*, Nov. 1902. *Neurol. Centralbl.*, Dec. 16, 1902, S. 1148.

THE writer here gives the results of his investigation of the mental condition of 120 vagrants who had been sent from the Workhouse to the Heidelberg Asylum. Most of them were old and many had previously been in prison. Of the 120 cases only 12 were women. As to the mental disease from which they suffered, there were 66 cases of dementia præcox, 19 of epilepsy, 7 of alcoholism, 3 of imbecility, 6 of hysteria, 4 of general paralysis, and four of paranoia. The 66 cases of dementia præcox were divisible into three classes, the first contained those who originally were normal in mind and body. Between 20 and 30 they had an attack of mental disease, and after an incomplete recovery from this they became vagrants. In the second class were included those who suddenly or gradually without assignable cause became restless and unsettled and gradually drifted into vagrancy. The third group was formed of those who all their life were eccentric and pathological, who even in early life showed intellectual and moral defects. These persons very early left home, wandered about, and, having no trade, took to pure vagrancy. The author does not state how many of his cases belonged to each of these classes.

JAS. MIDDLEMASS.

**BEITRAG ZUR PSYCHOLOGIE DER SITTICHKEITSVER-**  
(89) **BRECHEN.** ASCHAFFENBURG, *Versamml. mitteldeutsch. Psych. u. Neurol.*, Oct. 1902. *Neurol. Centralbl.*, Nov. 16, 1902, S. 1081.

By means of two tables based on government records of crime in France and in Germany the author shows the incidence of offences against morality for the various months in the year. They are shown to reach a maximum in June and July. He considers that a similar periodicity is demonstrable in all departments of sexual life. He further investigated the mental condition of all offenders against morality admitted to the prison at Halle for a year and a half and found that, in 80 cases, 60 showed some mental defect, while the rest were normal. In 37 there was imbecility of greater or less degree. In the great majority of cases the criminal tendency was traceable to youthful impressions.

JAS. MIDDLEMASS.

**LUNACY AND THE LAW.** By Sir WILLIAM R. GOWERS. Summary of an Address delivered at a General Meeting of the Medico-Psychological Association, Nov. 20, 1902.

SIR WILLIAM GOWERS, after some introductory remarks, said that the special subject which he desired to bring before them was the

harmful influence of the present Law of Lunacy in so far as concerned patients taken in for private treatment. The ostensible object of the law was the personal safety of the subject. To ensure this it is decreed that all persons of unsound mind shall be treated alike, certified as insane, deprived of liberty, and placed under the control of the Commissioners in Lunacy. No distinction is made as to the nature of the case, the needlessness or harmfulness of the proceeding. Thereby injustice and injury are done far exceeding that which the law can prevent. One criterion only is adopted—the technical evidence of mental unsoundness; one condition only determines its application—whether the care of the patient was paid for. The conditions are the same for the most harmless patient and the most dangerous. Yet any person, however violent, may remain uncertified in his own house, or under the care of those on whom he is dependent; but no other person, even a relative, may take a patient for payment without certification. Besides the many cases of mental unsoundness for whom the process of certification is needless and sometimes harmful, there is the large class of border-line cases, patients on the verge of insanity, some just over it. Many of them may recover, but they may be actually rendered insane by the process of being declared so, which certification constitutes. Such cases are very numerous. Three examples were mentioned. In one, a harmless delusion was the residue of a graver state, but made the patient technically unsound in mind. The law was broken by a doctor who received him into his house. In a fortnight the patient was quite well, and he has continued so now for six months. Had the law been complied with, the distress would certainly have greatly retarded the improvement, and perhaps would have prevented it altogether. In another case a harmless delusion prevented a patient obtaining a much needed change. An aunt who desired to take her could only do so on payment; she had been a nurse, and knew the law, and dared not run the risk. In a third case mentioned great strain in private life had recently brought a single woman to the verge of unsoundness, perhaps over it. She had an intense dread of going out of her mind. Under the care of a lady, who ran the risk of prosecution by taking her, she steadily improved. To have had her certified according to the law would probably have made her definitely insane.

It was well to consider what the process of certification is to the patient. The nearest relation must undergo the pain of signing a request that the patient shall be "detained and taken care off as a lunatic, idiot, or person of unsound mind." The last term is generally chosen, but it is well known to be synonymous with the first. Then follows an examination by two doctors, separately, who have, with such tact as they possess, to probe the inner secrets

of the mind and find out any delusion and the degree and character of any depression. Each has to make a declaration to the effect just mentioned. These documents are presented to a justice of the peace, who has power personally to examine the patient, happily not often exerted. Then follows removal to someone's care, a virtual imprisonment under the Commissioners until they release. The nature of the process cannot be concealed from many patients, and is most clear to those to whom it is most harmful. Too many on the brink of insanity are always haunted by the question "Shall I go mad?" To them it sounds the knell of hope, for it gives the answer, "You are mad." If the present law were strictly carried out it would cause a large increase in the number of the insane by destroying the chance of recovery which is often secured by breaking it.

It is a monstrous thing that the interests of the patient should be absolutely without influence in deciding whether certification should take place. That it is needless matters not; that it is harmful matters not. According to the law it depends solely upon technical evidence of mental unsoundness, upon what is essentially a legal point.

Why was this regulation made? The great fear was that the sane should be treated as insane, but this cannot be prevented by compelling all insane persons to be treated alike. It doubtless arose from a desire to guard against ill-treatment by placing all insane persons under the supervision of the Commissioners, but the danger of ill-treatment of those for whom there is payment is small. The cases of ill-treatment have been chiefly by those on whom the patients were dependent, and for these the law makes no regulation. Instances of ill-treatment of the weak-minded by those who received them for payment have been very rare, and of other forms of insanity almost unknown. A patient can leave or be taken away at any time. The present law actually does more harm than it prevents, and if strictly enforced it would do vastly more harm. That which constitutes the hardship is that it compels the compulsory certification of every case, however needless it may be, as a condition for the skilled care which can only be obtained for payment.

All the security the present law can give, and more, and all its harmful effects would be avoided by a system of notification. Let the law remain as it is for cases in which certification is necessary in the real interest of the patient. But for all cases in which this process seems unnecessary, and especially in early cases, in which there is so often a prospect of recovery, and all border-line cases, substitute the following system. Let everyone who receives such a case inform the Commissioners within a certain time. Let them, or someone deputed by them, visit the patient, and enjoin

certification, if necessary in the patient's interest. The visit could be repeated, and information should be given when the patient passed from care. But let the well-being of the patient, and the safety of others, be the only criteria. For justice' sake, for right's sake, abolish once and for all the artificial standard of technical mental unsoundness as determining the proceeding. It might involve an increase in the number of the Commissioners, but this was needed for other reasons. To give a large number of cases their best chance of recovery, the law must be constantly broken, with grave risk of prosecution to those who take charge of such. They feel that a sword is ever above them, hanging by what seems a thread.

The injustice of the present law is shown conspicuously by some of the prosecutions for its infraction, for the "illegal charge of cases," which the Commissioners are obliged to undertake. Several examples of these were given from the reports of the Commissioners. In one, an old lady was bedridden from paralysis of all her limbs, due to brain disease, which had also caused delusions. She was well cared for in the house of a doctor. Information was received by the Commissioners, perhaps from some discharged nurse, and the doctor was prosecuted, convicted, and fined. The unhappy lady, at the instigation of the Commissioners, was certified as a lunatic, removed, and placed under other care. Again, a lady in a nursing home at York had to be certified and moved to an asylum. She had been in the home for three months in the hope that the treatment there might do good. In consequence the lady manager of the Home, and the nurse also, (though there was no allegation of ill-treatment), were prosecuted, convicted, and fined. A patient was received by a lady in an East coast town who, in a few days, wrote to the friends that the case was too serious for her, but, because a month elapsed before the patient could be transferred under certificates to an asylum, the lady was prosecuted. The Justices seem to have had no choice but to convict, although their common sense prevailed, and they only told the lady to come up for judgment when called on. In other cases of prosecution there was no pretence of need, so far as the patient was concerned, but a professional opinion of technical mental unsoundness ensured conviction.

In Scotland the law is far more reasonable and humane ; any patient, either on the verge of insanity or definitely insane, can be taken uncertified for six months, "with a view to recovery," on a simple medical recommendation. The system answers well, and prosecutions seem unknown.

How vast is the work of the medical Commissioners the Reports show. They have their own responsibility for all the insane under their supervision. They are three, the same in



number as when they were first ordained in 1845. The number of the insane under them is not known before 1859, it was then 36,700. Now it is 110,700 ; so it is not likely in 1845 to have exceeded 27,000. There would be then one medical Commissioner to 9000 cases, and now there is one to 36,000. If the original proportion was right (and more supervision is exacted now than then), there should, at the present time, be twelve medical Commissioners instead of three. In Scotland there are now two for 15,800 insane, and the same proportion would involve fourteen for England and Wales. Doubtless the work of the three legal Commissioners has correspondingly increased, but it is less in evidence. Besides the six Commissioners there are five other legal members of the Board which presides over this vast department of disease. Surely these are grounds for a thorough examination of the work and organisation. Other subjects also press for consideration. The need for hospitals to receive border-line cases has often been urged, and is unquestionably great. The time has certainly come when a Royal Commission should investigate both the work and constitution of the Board of Lunacy, the working of the Lunacy Act, some remedy for its unjust and harmful effects, and the need for other provisions than those it affords. The time—twelve years—which has elapsed since that Act was passed has furnished ample experience of its effect and deficiency. But a thorough investigation must of necessity take time. Meanwhile, should this hardship be unrelieved? It was earnestly to be desired that as a temporary measure either the Scotch system should be made legal, or, what would perhaps be simpler, the following brief enactment should be passed—so obviously right and just that it could scarcely meet with opposition—an enactment that “the provisions of the Lunacy Act relating to private patients taken for payment should apply only to such cases as, in the judgment of the Commissioners, need to be certified and detained in their own interest or for the safety of others.”

The head of the Board of Lunacy is the first lawyer of the kingdom, and at the present time he is such not only by position but in reality, and is one who has also a sense of what is right and just so keen that an adequate appeal to him cannot be in vain. Vast and multifarious as is his work, so great is the trust in the Lord Chancellor that the Lunacy Act was passed through both Houses of Parliament in 1890 without discussion, as Hansard shows. But even the Lord Chancellor is beneath the law, and grave indeed is the responsibility of the Legislature. If its members, having eyes, saw not what was outside their range of vision, they were free from blame ; but if, having ears, they now hear not what is testified by those who see, is not their condemnation written ?

Finally, Sir W. Gowers said that he had taken the opportunity of bringing the subject forward under a compulsory sense of duty. He might fail, and indeed could succeed only by arousing the efforts of others, but failure could not be for long; no grave injustice, once perceived, remains long unredressed.

AUTHOR'S ABSTRACT.

### TREATMENT.

**LE TRAITEMENT DES PSYCHO-NÉRVOSSES À L'HÔPITAL PAR**  
(91) **LA MÉTHODE DE L'ISOLEMENT.** Par J. DEJERINE,  
*Rev. Neurolog.*, Dec. 15, 1902, p. 1145.

SINCE 1895 Dejerine has treated two hundred cases of hysteria, neurasthenia, and hystero-neurasthenia in the wards of the Salpêtrière, by complete isolation and milk diet, somewhat on the plan advocated long ago for private patients by Weir-Mitchell, but without massage. During this time he has had only two failures, and he finds that the recovery is more complete and more rapid than with private patients, and that this plan is also very applicable to patients who have been previously treated unsuccessfully in hospital wards without isolation. Along with the isolation milk is given every two hours, and rapidly increased to about five or six litres in the day. An essential part of the treatment is the gaining of the patient's confidence, the explanation to her of the nature of the symptoms, and firm yet kindly discipline, with the encouragement that the rigour of the isolation will be relaxed in proportion to the amelioration of the symptoms.

Dejerine finds that the treatment is suitable to the most severe cases of hysteria, neurasthenia, hystero-neurasthenia, mental anorexia, and intractable vomiting, and he has been surprised with the rapidity with which the symptoms have yielded. He states that the symptoms which characterise what might be called "major hysteria" have never lasted for more than a week after the commencement of the treatment, and as the ultimate result he has obtained in all his cases, not merely an amelioration, but an actual recovery.

The reviewer can speak from personal experience of the merits of this treatment, which deserves to be more widely known and practised.

ALEXANDER BRUCE.

## Review

**DIE SYRINGOMYELIE.** Eine monographie von Dr HERMAN (92) SCHLESINGER, Privatdocent aus dem *Neurologischen Institute an der Wiener Universität*. Zweite vollständig umgearbeitete und bedeutend vermehrte Auflage, mit 88 Abbildungen im Texte. Franz Deuticke, Leipzig und Wien. 1902.

THIS book affords a striking instance of the rapid strides which have been made in the science of Neurology during the closing years of the nineteenth century. Twenty years ago, it was shown by Kahler and Schultze that the pathological condition which had been named by Ollivier (1827) Syringomyelia might in some cases be diagnosed during life. In the period which has elapsed since this discovery, contributions to the study of the disease have been very numerous. In 1894 Schlesinger produced an elaborate monograph on syringomyelia, which at once took its place as a classic in medical literature. The exhaustive treatise before us is a new edition of that work greatly enlarged and in many places entirely rewritten.

The author's experience of the disease has been very large, he has personally observed no less than 120 cases during life and has had 30 post-mortems. The book is based not only upon his own personal experience, but also upon a bibliography of 1175 items, most of which he has had the opportunity of reading in the original (see p. 574).

The opening chapter is devoted to an interesting historical sketch in which the gradual steps in the evolution of our knowledge of the disease are traced from the time of Etienne, who in 1564 described a spinal cord with a cavity in its substance, up to the present date.

The author in a few sentences defines syringomyelia before proceeding to a description of its clinical features.

The symptomatology of the disease is treated under six main headings, viz.: motor symptoms, sensory symptoms, trophic changes, the condition of the reflexes, disturbances of the genito-urinary system and rectum, and symptoms referable to the medulla and higher parts of the brain. This description occupies almost two hundred pages.

The very various types under which the disease may present itself are then considered and individually described. This classi-

fication appears to us of such importance from the standpoint of diagnosis that we offer no apology for quoting it in full.

Chief types of syringomyelia:—

1. Syringomyelia with classical symptoms.
  - (a) Cervical type.
  - (b) Dorso-lumbar type.
  - (c) Sacro-lumbar type.
  - (d) Bulbo-medullary type.
2. Syringomyelia with preponderating motor symptoms.
  - (a) Resembling amyotrophic lateral sclerosis.
  - (b) " spastic spinal paralysis.
  - (c) The scapulo-humeral type.
3. Forms with predominating sensory phenomena.
  - (a) Forms simulating hysteria.
  - (b) General anæsthesia.
4. Syringomyelia with preponderating trophic disturbances.
  - (a) Syringomyelia with Morvan's symptom complex.
  - (b) The osteo-arthritic type.
5. The tabetic type.
6. The pachymeningitic type.

The differential diagnosis from the various diseases which may resemble the above types is very fully considered. The author points out that hysterical symptoms are not uncommonly associated with syringomyelia and may form a prominent feature of the clinical picture. The differential diagnosis between syringomyelia unassociated with tumour growth and cases of intra-medullary tumour may be very difficult, but is of great importance. The average duration of life in 62 cases of intra-medullary tumour was 20 months, whereas patients with syringomyelia often live as many years. No single symptom of syringomyelia is pathognomonic, and a diagnosis is not justifiable in the presence of only one of the cardinal symptoms of the disease.

Schlesinger's experience as to the frequency of syringomyelia is of interest. He states that in his opinion "syringomyelia is one of the most common of the spinal cord diseases and ought as regards frequency to follow closely tabes and the syphilitic and pressure paraplegias." On the same page he remarks that syringomyelia is more often met with than disseminated sclerosis. It would be interesting to know whether this is also the experience of other continental observers.

A chapter is devoted to the question of a possible relationship between leprosy and syringomyelia; the author sums up by agreeing with Laehr that the two diseases are perfectly distinct and depend upon a different pathology.

The prognosis as regards life in cases uncomplicated with tumour is distinctly favourable, although patients with syringo-

myelia are always liable to hæmorrhage into the cord, occurring either as the initial manifestation or in the later stages of the disease.

A description of the pathological anatomy of syringomyelia including a chapter on the normal ependyma and the neuroglia of the central canal occupies 100 pages. The conditions in which cavity formation is met with in the cord, viz.: pure hydromyelia, primary gliosis with syringomyelia, and cavity formation in tumours, in spinal pachymeningitis and after trauma and hæmorrhage, are described in detail.

The metamorphosis of the glia is according to Schlesinger closely related to hyaline and mucoid degeneration, and yet it is not identical with these nor with colloid or amyloid degeneration; the word "*homogenisation*" is suggested by the author as most descriptive of its chemical properties. With regard to the changes often met with in the posterior columns, in the great majority of cases they occupy the ventral region of the columns.

Referring to the pathogenesis of the disease, the author states his opinion that the anatomical etiology of syringomyelia is not uniform. In rare cases, syringomyelia may be the product of a chronic myelitis with or without congenital changes. This is, however, only a possibility which has not yet been definitely proved.

In the last hundred pages of the book, the details of 56 cases of syringomyelia are recorded.

An exhaustive bibliography which has been already referred to completes the work.

Numerous illustrations are introduced in the text.

The book is one which all neurologists should possess. We venture to think that it will remain the standard work on the subject for many years to come.

EDWIN BRAMWELL.

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# Review of Neurology and Psychiatry

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## Original Articles

### ON THE LIP-REFLEX (MOUTH PHENOMENON) OF NEW-BORN CHILDREN.

By JOHN THOMSON, M.D., F.R.C.P.Ed.,  
Physician to the Royal Hospital for Sick Children, Edinburgh.

IN 1896, while making a series of observations on Chvostek's symptom (facial irritability) in infancy, my attention was drawn to the existence of a normal reflex movement of the lips in sleeping babies, which occurs on tapping near the angle of the mouth. Since then I have investigated it further, and have examined some hundreds of children of various ages as to its presence. The thing is so easily observed that it must be familiar to many who have to do with young infants; yet I have only been able to discover two very short accounts of it in medical literature (Loos, Escherich). I have been in the habit of calling it the "lip-reflex," and I am still inclined to prefer that name as being more distinctive than the term "mouth-phenomenon," which Prof. Escherich uses.

When so much is being written on various other minor reflexes, this small phenomenon seems to deserve more study than it has as yet received. A few facts about it are therefore, perhaps, worthy of record here.

#### *Mode of Eliciting the Reflex.*

The reflex is best elicited by a series of gentle taps on the upper lip a little above the angle of the mouth, or on the under

lip a little below it (Fig. 1).<sup>\*</sup> It can, however, be got anywhere on the lips in a well-marked case, and sometimes over a considerable part of the cheek. A gentle touch on the mucous membrane of the lips, such as might be given by the mother's nipple, will often originate some degree of the movements. Loos refers to Chvostek's point and the region over the sucking pad as the places to tap in order to start the reflex. I have not, however, succeeded in eliciting it from these situations nearly so often nor so readily as from the lips.

The taps may be given by the finger-tip alone; but they can be more accurately directed if a light stiff pointed object is attached to the end of it. For this purpose a small-sized vulcanite ear-speculum, with its extremity padded, acts very well; and it can easily be attached to the finger-tip by means of an elastic band.

*Description of the Phenomenon.*

On tapping the upper lip, in a well-marked case, there is often, first of all, a slight momentary jerk. This is generally towards the side tapped but sometimes towards the other side. Almost at the same time the lips close, if they have been parted, and become deliberately pursed together so as to pout a little. As the tapping is repeated, the protrusion of the mouth becomes more and more marked (Fig. 3). In some instances the projection is straight forward, but generally the central point of the mouth turns markedly towards the side *opposite* to that tapped. Both upper and lower lips participate in the pouting. In some cases the preliminary jerk is not seen. In some, after repeated tapping, there are to and fro sucking movements of the tongue along with the pouting. When the lower lip is tapped, the resulting phenomenon is much the same.

*Circumstances under which it occurs.*

The lip-reflex seems to occur more or less distinctly in all healthy new-born babies when they are sound asleep; and in a considerable proportion of them when they are sleeping lightly, or even are only drowsy. I have seen it in a few new-born infants who were evidently quite wide awake; but this is rare.

<sup>\*</sup> The illustrations represent an infant of twelve weeks who was taking chloral for convulsions. They are from photographs kindly taken for me by Dr H. O. Nicholson.

THE LIP REFLEX.



FIG. 1.



FIG. 2.



FIG. 3.

Fig. 1. Before tapping. Fig. 2. After first tap. Fig. 3. After several taps.



As the children grow older the reflex is less frequently found, and is only present when they are sound asleep. Until the end of the third or fourth year it is fairly common. After that it is less so, and it is usually also less marked in character. I have found it in older children up to twelve years, but have not examined for it in adolescents or adults.

Infants of a few months who are taking large doses of chloral on account of repeated convulsions often show the lip-reflex in a peculiarly marked degree. Probably this may be due merely to their being able to stand an unusual amount of percussion without being roused by it from their deep sleep. I have, however, seen it very strongly present in two babies who were taking convulsions and who had not had any sedative drugs.

#### *Nature of the Lip-Reflex.*

Throughout this paper I have referred to the phenomenon as a reflex. That it is a true reflex and not merely the result of direct stimulation of the nerves or of the muscle can scarcely be questioned. The following facts with regard to it seem to bear this out :

1. The greater part of the characteristic movement is deliberate, co-ordinated and quasi-purposive.
2. Repetition of the tapping has a distinctly cumulative effect—the protrusion of the mouth never reaching its maximum until the lip has been stimulated over and over again.
3. Both sides of the mouth move in response to tapping on one side, although often not to an equal extent; and both lips act when one is touched at any point.
4. However markedly the lips move in response to tapping while the child is asleep, they nearly always cease to do so at once whenever he awakes.

Chvostek's symptom differs widely from the lip-reflex. It, of course, is not of the nature of a reflex at all, but is due to mechanical stimulation of the facial nerve at the point of impact. Unlike the lip-reflex it is probably always to some degree pathological in its significance. It occurs equally during sleeping and waking. The movements which it exhibits are confined to the side tapped; and in character they are momentary, not co-ordinated and non-purposive.

The symptom which is most likely to be mistaken for the lip-reflex is that which Thiemich has described as the "lip-phenomenon." In this, a tap on the upper lip is followed by a sudden contraction of the orbicularis oris which produces a momentary protrusion of the mouth. I have seen many instances of this. It occurs in children who are wide awake, and is sometimes very well marked. From the character of the phenomenon, the conditions under which it is met with, and the subsequent history of the children who show it, it seems almost certain that it is merely a localised variety of Chvostek's symptom.

An infantile reflex act which presents some analogies to the lip-reflex is the normal extensor response which occurs on tickling the soles in babies. The extension of the great toe, spreading out of the other toes, eversion and dorsiflexion of the foot with drawing up of the leg constitute a somewhat similar co-ordinated quasi-purposive act. This also ceases to occur, in the same form at least, as the child grows older, but it is still occasionally observed during deep sleep in bigger children who are quite healthy.

When we consider that the act of sucking is the most fully developed voluntary co-ordinated act of which the newly-born child is capable, it is not surprising that the lips should be the seat of a special reflex at birth. It is evident that the lip-reflex serves a very useful purpose in assisting the infant's first unpractised attempts at sucking. By its means the mouth assumes automatically a more convenient shape for receiving and retaining the nipple. At the same time it is interesting to remember that the mother's nipple is provided with a corresponding reflex. When it is mechanically stimulated its muscular fibres contract in such a way that it becomes harder, longer and thinner; it is thus more easily grasped and retained, by the infant's mouth.

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## CONGENITAL FACIAL DIPLEGIA DUE TO NUCLEAR LESION.

By HARRY RAINY. M.D., F.R.C.P.Ed., and J. S. FOWLER,  
M.D., F.R.C.P.Ed.

WE are led to report this case for two reasons: (1) It belongs to a definite though somewhat rare group of cases known in Germany as *infantile Kernschwund* or *Muskelschwund*, of which ophthalmoplegia externa, with or without involvement of the other cranial nuclei (or the muscles innervated from them) is the most common variety, and although it has been conjectured that cases of facial diplegia alone belonging to this class might occur, none, so far as is known to us, have been reported up to the present time.\* (2) The disease is not in itself fatal, and of the very few pathological examinations made, nearly all have been in adults in whom the condition has lasted for many years. The youngest patient in whom the nervous system has been microscopically examined is a child of two and a half, carefully reported by Heubner.

*Clinical History.*—Mary B., aged ten weeks. Brought to the out-patient department of the Sick Children's Hospital on May 21st, 1902. *Complaint.*—Difficulty in swallowing, face remains motionless when the child cries, and the lower lip is drawn in at the one side. *Duration.*—Since birth. *History.*—Family history negative. The patient is a first child, and labour was instrumental, the forceps leaving marks on the forehead. The infant was large and healthy at birth, but very cyanosed, and required artificial resuscitation. The child was put on the breast soon after birth, but refused to suck. She took very little food for the first fortnight and got much thinner, and eventually had to be fed with a spoon. When food was introduced into the mouth she seemed to be able to swallow fairly well. Though she at first vomited a good deal she has never returned food through the nostrils. Bowels are constipated; there has never been any diarrhoea.

Some time after birth it was noticed that when she cried the

\* Since the above was written Dr Seymour Taylor (Clinical Soc. Trans., vol. xxxv., 1902) has reported a case of congenital facial diplegia in a boy of fifteen, complete, except for slight movement of the risorius, depressor anguli oris, and orbicularis oris, which is probably of the same nature as the case under consideration.

face remained motionless with the exception of the right side of the lower lip, which was drawn in a little. Nothing abnormal has ever been noticed as to the movements of the eyes or eyelids. The child sleeps well, and has had no otorrhœa and no convulsions.

*State on Admission.*—Height 20 ins., weight 8 lbs. 2 ozs. A poorly nourished infant with a dry scaly eruption on the arms and scalp. The cry is feeble, and there is slight snuffling. The head measures  $13\frac{1}{2}$  ins. in circumference, and the cranium is of normal shape. The anterior fontanelle is about 1 in. in diameter; there is neither craniotabes nor facial irritability. Pulse 138, temperature  $96^{\circ}$  F. The abdominal viscera are normal. Respiration is costo-abdominal, 47 per minute; slight bronchial catarrh is present. The circulation is normal.

*Nervous System.*—There is complete paralysis of both seventh nerves and the face is practically motionless and devoid of expression when the child cries, except that there is (*a*) a contraction of the fibres of the occipito-frontalis just above the extreme inner angle of the right eye, and (*b*) of the right depressor anguli oris. The upper lip protrudes somewhat, while the lower one falls in and is drawn slightly downwards and towards the right side. On putting the finger into the mouth the lips hang loosely round it, and are pushed in before it, making no effort to close on it, though when it enters the mouth the jaws can be felt to clench and sucking movements of the tongue take place. The eyes are not closed very tightly or completely, especially on the left side. (At a later period of the child's stay in hospital, however, the eyes were fairly well closed during sleep.) The movements of the eyes are normal in all directions, but sluggish, apparently owing to the general apathy. Swallowing is quite well carried out when food is put into the mouth by a tube. There is no other paralysis. The knee jerks are difficult to elicit. Taste is present, but whether over the whole tongue or not is uncertain. The *electrical reactions* were determined on two occasions. On the first of these the masseters and orbiculares palpebrarum responded feebly to both the faradic and the galvanic current. No other facial muscles responded to either. On the second examination there was absolutely no response on the part of any of the muscles supplied by either seventh nerve to any form of stimulus.

The child died a few days after admission.

As regards the seat of the lesion in this case, the opinion formed was that it was probably nuclear. The diagnosis (which was, as will be seen, confirmed by subsequent examination) was based largely on the feeble electrical reaction and absence of R.D., and subsequent total absence of response to electric stimulation, which could scarcely have been the case had the facial palsy been due a peripheral forceps trauma at birth ten weeks earlier. While unilateral facial palsy is a common accident in forceps cases, the bilateral form is almost unknown, as might indeed be expected on account of the improbability of the forceps being so applied as to press on both facial nerves. The only case of this kind which we have found in the literature is that of Edgeworth (1), in whose patient, a girl aged seven, there was a clear history of bruising in front of each ear after forceps delivery. There was complete paralysis of the upper branches of both facial nerves, and partial paralysis of their lower fibres. The fact that in our patient there was slight movement of the muscles about the right eye and right angle of the mouth, was also in favour of the diagnosis of central lesion.

The infant died of broncho-pneumonia, and the post-mortem appearances corresponded to the condition observed during the last few days of life. The brain was carefully removed; it presented no naked-eye indications of disease, and was at once prepared for microscopic examination by Marchi's method. Portions of both temporal bones, containing the seventh nerves, were also taken. These were placed in Muller's fluid and the nerves were subsequently dissected out. The facial muscles were found to be greatly atrophied, but a few portions were identified and fixed for histological investigation.

The following are the results of the examination of these tissues.

#### I. THE BRAIN.

*Method.*—Sections were prepared and examined from the second cervical segment to a point above the third nucleus. In the regions where there was any ground to believe that changes might have occurred, at least every tenth section was examined; and in the region of the seventh nucleus and nerve root every section was mounted, so that a complete series of this region was available. In some of the sections Marchi's reaction was sup-

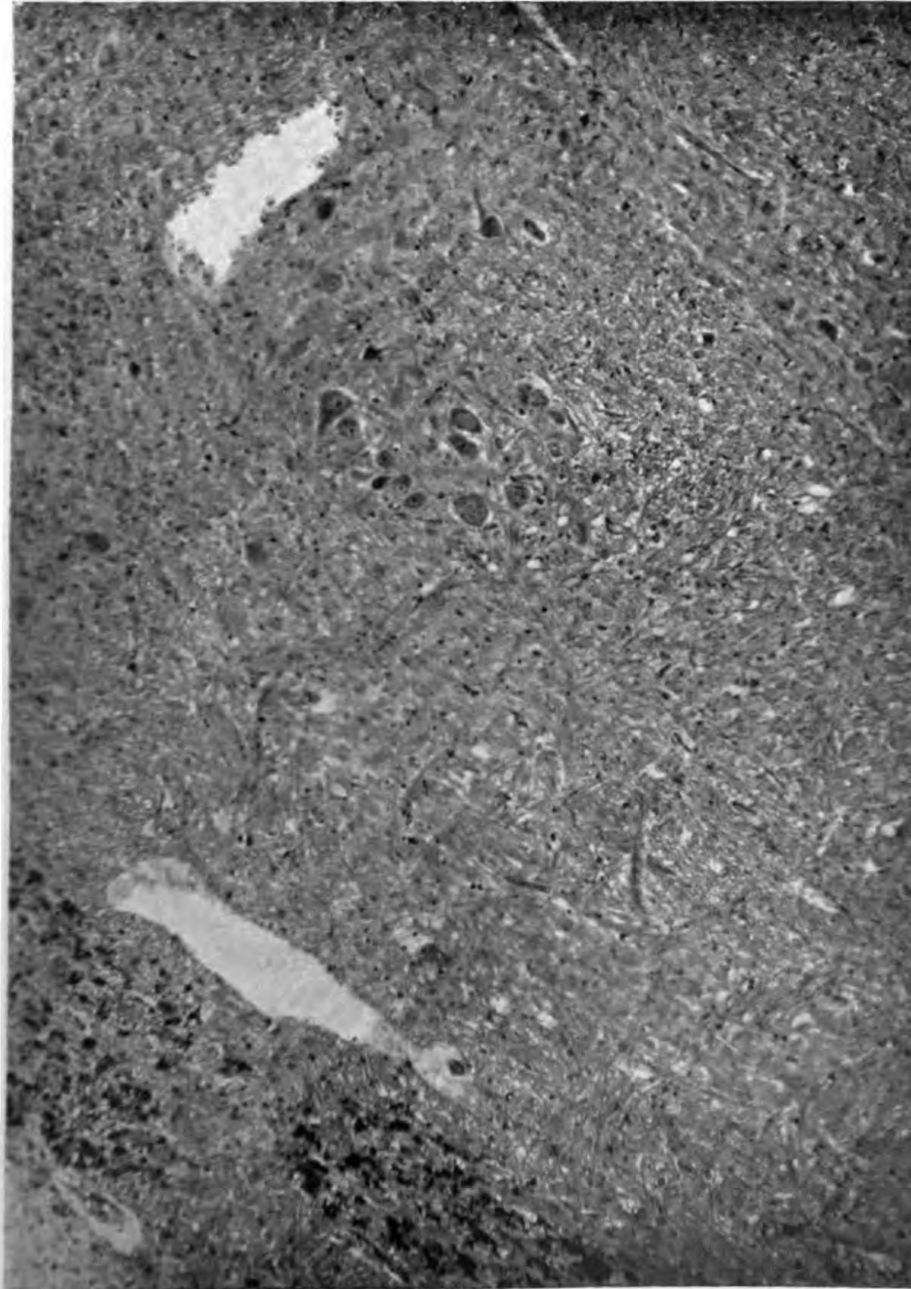
plemented by counterstaining in order to show details of cell structure. The stains which we found most suitable for this purpose were van Gieson's and picro-fuchsin, and with these, in spite of the fact that Marchi's method of fixation is unfavourable for the best rendering of detail in nerve cells, we succeeded in securing sufficiently clear pictures.

*Results.*—Except in the seventh nerve and its nucleus no abnormality was detected. There were, indeed, some black deposits in several other nerves, especially in the sixth pair, but not more than is commonly seen when some hours have intervened between death and their fixation, and their nuclei showed no indication of disease or atrophy. In the case of the seventh pair the condition was totally different (Plate 2). The nerve on both sides showed very marked degeneration, alike in the ascending part of the root, in the fasciculus teres, and in the emergent portion. Degenerated fibres could also be clearly traced passing through the healthy sixth nucleus in the manner described many years ago by Gowers and von Gudden. Careful scrutiny failed to recognise any of the fibres which Bruce has described as crossing the middle line to enter the opposite nerve, and we could not detect degenerated fibres at any level in the posterior longitudinal fasciculus. This last point is of some interest in relation to the supposed entrance into the seventh nerve of elements extraneous to its own proper nucleus, although it is inconclusive, since degenerated fibres would only have appeared in the event of their cells of origin having perished.

The condition of the nuclei of the seventh pair was very interesting. A large number of the cells ordinarily present had disappeared, whilst those which remained presented a distinctly atrophied aspect, their processes being ill developed, the Nissl's bodies irregular, and the cells themselves much smaller than one would have expected. The apparently healthy cells of the sixth nucleus, which were present in many of the sections, afforded a marked contrast to those of the seventh, which still remained, and established the fact that the appearances noted in the latter were not due to faulty technique in preparing the sections.

## II. THE FACIAL NERVE TRUNKS.

*Method.*—After fixation in Muller's fluid the nerves were divided into suitable lengths, some of which were cut transversely



*Transverse Section through Medulla at Level of Seventh Nuclei.*

To the left a portion of the emergent root of the seventh nerve is seen crossing the section diagonally. It is deeply stained by Marchi's method. A little to the right of the middle of the plate are a few of the remaining cells of the seventh nucleus.



and others longitudinally. The sections were then stained in various ways, including Weigert Pal's, van Gieson's, and Ehrlich's triple stain.

*Results.*—All these methods showed advanced degeneration of the nerves, and fully confirmed the results which had been yielded by the Marchi staining of the nerve roots.

### III. MUSCLE.

The few fragments of muscle which could be found appeared normal when submitted to histological examination. Probably they were associated with the few surviving cells and nerve fibrils of the seventh pair.

Möbius (2) was among the first to draw attention to the condition of which this case is an example, and his classification of the cases and description of their symptoms have practically been accepted by subsequent writers. He describes (1) Ophthalmoplegia externa, congenital, or beginning in childhood or youth, with which may be associated facial paralysis. (2) Bilateral abducens paralysis, with or without facial palsy. (3) Bilateral paralysis of the third nerves. (4) Bilateral ptosis. (5) Unilateral ptosis and abducens paralysis.

These cases have the following features in common: (1) They usually are congenital or occur in youth, and when the symptoms attain a certain maximal development they remain stationary during the rest of life. Most of the recorded cases had already reached this stage at the time of observation. (2) The external ocular muscles are often involved; the iris and ciliary muscle never. Along with this, the facial or hypoglossal nerves, and perhaps certain spinal nerves, may be affected. Möbius conjectured that facial and hypoglossal palsy of this nature might exist alone, though at the time of writing no cases were known to him, and subsequent events have justified his supposition. (3) The paralysis is purely motor; there is a total loss of all electric excitability, and never reaction of degeneration. There may be actual absence of the muscles supplied by the nerves involved. (4) The condition is nearly always bilateral and symmetrical. (5) While there are no other nervous symptoms of any kind, developmental anomalies, chiefly of a trivial nature, are not uncommon.

*Nature of the Condition.*—Möbius argued that the disease

might either be allied to the muscular dystrophies or of primarily nervous origin, and concluded on clinical grounds that it was due to degeneration of the nuclei. Subsequent writers, in particular Heubner (who was the first to describe the pathology of a recent case), while agreeing that the condition is of nervous origin, think that the change is not a degeneration, but a hypoplasia or an aplasia of the nerve centres concerned. Heubner's case (3) was one of bilateral abducens paralysis, total left and partial right facial paralysis, and atrophy of the left half of the tongue in a child of two and a half, the condition dating from birth. The nuclei of the sixth, seventh, and twelfth nerves were small and the cells few in number, while the left olive and pyramidal tract were also poorly developed. Recent degeneration of the nerves was not demonstrable by Marchi's method, and neither the nuclei nor the muscle fibres showed signs of degeneration. The facial nerves were not examined except in their course through the pons.

Nothing is known as to the cause of the condition. In only a few instances has it been hereditary.

While the pathological appearances in our case are conclusive as to the central origin of the paralysis, they are opposed to Heubner's suggestion that the condition is one of aplasia or hypoplasia of the implicated parts, and our reasons for making this statement are as follows :

- (1) Since degenerated nerve fibres are present there must at an earlier stage have been nerve cells from which they took origin ; and, therefore, aplasia of nerves and cells is absolutely excluded. Probably Heubner's case, in which the condition had lasted for two and a half years, was one of too long standing to permit of the application of Marchi's method with any prospect of obtaining positive results.
- (2) Since the electrical reactions (so far as an opportunity was available for observing them during the few days when the infant was in hospital) appeared to fade out without the occurrence of any reaction of degeneration, one must conclude that no sudden accident, whether of the nature of a traumatism or an acute polioencephalitis, abruptly severed the muscles from their nutrient centres, whilst the bilateral symmetry of the lesion is also adverse to such an opinion being maintained.



The conclusion, therefore, to which this case clearly points is that, either through lack of inherent vitality, or through defective nourishment, or through the gradual action of some noxious influence which may have been of a toxic nature, the cells slowly perished, leaving evidence of their former existence in the degenerated nerves and the atrophied muscles which they formerly controlled.

In closing, we have to thank Dr John Thomson, under whose care the patient was, for kind permission to record the case, and Dr Noël Paton for giving every facility for the histological examination of the specimens at the Laboratory of the Royal College of Physicians.

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### NOTE ON SYRINGAL HÆMORRHAGE INTO THE SPINAL CORD.

By SIR WILLIAM R. GOWERS, M.D., F.R.S.

CASES of sudden or rapid disease of the spinal cord are sometimes met with which present peculiar symptoms which make it probable that the lesion is hæmorrhage into a congenital cavity. Their distinctive symptoms are the prominence and definite character of sensory paralysis combined with motor palsy. Such cases are rare, but it is possible that less definite symptoms may be produced by a similar lesion and that it may not be so rare as the cases with defined symptoms suggest.

Syringal hæmorrhage is not an hypothesis. In a spinal cord figured in my "Manual Dis. Nerv. System" (vol. i., Fig. 188, 3rd edition) a cavity, evidently congenital from its limitation by embryonal tissue, was filled with blood from the cervical region to the lower part of the cord. It passed from the central canal backwards on the inner side of the posterior horn. Through part of the dorsal region a similar smaller cavity existed adjacent to the right posterior horn, in which there was no blood. But the manner in

which the cervical cord, at the seat of the chief extravasation, was torn up, and distended with blood, suggests that the hæmorrhage had begun here, and after distending the cavity to the lowest part of the cord, has spread in the substance of the cord at its place of origin. This is also suggested by the history of the case. Such cavities, with the embryonal tissue adjacent, must entail lessened support to the vessels, and facilitate the occurrence of hæmorrhage. Adjacent gliomatous new formation must entail increase vascularity. It is possible that some of the cases in which these cavities have been thought to be caused by hæmorrhage were really congenital, into which a vessel had ruptured.

True syringal hæmorrhage is probably seldom fatal. The blood may escape into the cavity, which is distended, and symptoms are due to the compression of adjacent structures, and not to their destruction. The cavity constitutes a receptacle for the blood, limited by its extent. This is indeed hypothetical, but it is reasonable, and it explains the absence of pathological proof of the nature of such cases as the following :

Case 1. Dr C., aged 40, motor and sensory palsy of the left arm, came on in the night during sleep. There was no affection of face or leg. Two months afterwards there was complete paralysis of the arm and also of the sternomastoid, trapezius and pectoralis, with slight wasting, and only slight diminution of electrical excitability of the muscles, similar to faradism and voltaism. Sensibility was lost, in all forms, over the arm and neck up to the edge of the jaw and over the side of the thorax down to the edge of the ribs. In the leg there was only a greater knee-jerk than on the other side. The symptoms continued for two years, and then slowly lessened. Now, four years after the onset, there is only slight weakness of some movements of the arm, and slight diminution of sensibility in the upper arm, shoulder, and over the scapula.

Palsy of such distribution could only be spinal. Its onset suggests hæmorrhage, and its limitation implies that this may have been into a pre-existing cavity. Otherwise the escape of the right arm is inexplicable. This pathology was confirmed by another symptom. From birth there had been inability to move either eye to the right of the middle line, and almost complete absence of movement of the left eye. This could only be due to a congenital defect of structure on the left side, such as might well be connected with a cavity on the same side of the cord. Although

there were no leg symptoms, a morbid state of the hip-joint led to its resection by Mr Symonds at Guy's Hospital. The head of the femur was greatly enlarged and was sawn off. The edges of the acetabulum was also much thickened. Although ascribed to a dislocation at 2, the condition resembled "tabetic arthropathy," a known effect of syringomyelia. He had phlebitis in the leg after the onset of the palsy and also after the operation, which is said to have the same association.

Case 2. A man, aged 41, a subject of the hæmorrhagic diathesis. At 16 he jumped over a box and the next day was paraplegic with loss of motion and sensation. He slowly regained motor power, but sensory defect remained, and 24 years afterwards sensation to both touch and pain was absent over an area on the backs of the thighs, the lower legs and feet, corresponding to the area corresponding to the fifth lumbar and all the sacral segments, with the exception of a small area on the outer side of the left foot which was hyperaesthetic. Otherwise the bilateral symmetry of the loss was perfect. The only residual motor symptom was some weakness of the flexors of the ankles. Such symptoms suggest a lesion determined in area by pre-existing structural conditions, and the escape of blood into symmetrical cavities, connected at the central canal, affords the best explanation. The hæmorrhagic diatheses may well have rendered a concussion effective. Additional evidence was afforded by a condition of the knee-joints resembling tabetic arthropathy, with much thickening of the adjacent ends of the bones. There was also chronic change at the hip-joint. He had also suffered from an obstinate recurring ulcer on one toe.

I have seen some other cases, suggestive of syringal hæmorrhage, although less strongly. One of them was instructive because symmetrical sensory loss on the legs passed away more slowly than motor palsy, and power was regained without co-ordination.

The two cases suggest that syringal hæmorrhage is the probable lesion in cases of sudden spinal palsy in which sensory loss is well defined and persistent, presenting a limitation unusual in random lesions, either unilateral, or bilateral and symmetrical. The cases also suggest that the ultimate prognosis in such cases is better than the gravity of the symptoms would indicate.

I have discussed the subject in a recent lecture which will shortly be published.

## CLINICAL OBSERVATIONS IN ACUTE CONTINUOUS MANIA

By LEWIS C. BRUCE, M.D., Phys. Supt., Murthly Asylum

IN the classification of mental diseases there is much confusion; each country has a classification of its own, and one might almost add that each authority or specialist has also a classification of his own. Where there are no settled beliefs there can be no foundation of clinical facts. I have now therefore to explain what I mean by the term "acute continuous mania."

The majority of writers in writing of "mania" treat it at one time as a symptom and at another time as a distinct disease with many varieties, viz., puerperal mania, epileptic mania, alcoholic mania, etc., and they further describe "mania" as a distinct disease, and divide it into "simple mania" and "acute mania," and there is no attempt made to differentiate the symptoms of this disease, acute mania, from the symptoms of manias which complicate puerperal insanity, epileptic insanity, etc.

The disease which I now describe as "acute continuous mania" is probably the same disease described by most authors as "acute mania." I believe it to be a condition of acquired toxæmia resulting in definite physical and mental symptoms, which differentiate it—not very markedly I must admit—from the manias of adolescence, gross brain lesions, senile decay, the puerperium, the epileptic, the alcoholic, the general paralytic and the subject of folié circulaire.

The onset of acute continuous mania is as a rule gradual. The patient feels out of sorts, is generally sleepless, restless and unable to attend to business, or concentrate the mind upon any work for any length of time. The physical symptom, which is almost invariably present, is dyspepsia with flatulence. The maniacal attack itself may be gradual or sudden in onset; self-control is lost and the patient is then certified and placed in an asylum. In the history of the patient one finds frequently hereditary predisposition to insanity. Secondly, it is not uncommon to find that the patient has exceeded in alcohol shortly before the onset of the illness. In such a case alcoholism

is a symptom, not a cause of the disease. It is also common to find that the patient has passed recently through a period of intense mental worry or anxiety, often with bodily privation or unhealthy environments. Some condition, in short, has occurred which has lowered the natural resistive powers of the patient.

The patients who suffer from this type of disease are by no means weaklings. Although I have seen the disease occur in adolescents, as a rule it attacks adults, and in the great majority of cases these patients are well developed, although they may show want of condition. The face is drawn and pale, the eyes bright and staring with widely dilated pupils. The temperature varies according to the stage of the attack. At the onset the temperature is rarely febrile, although I have seen an occasional rise to  $99^{\circ}$  in one or two patients more than usually excited, but these are exceptions to the general rule. This symptom alone assists in the diagnosis between mania and acute melancholia in the early stages, and also between mania and confusional insanity, a disease often mistaken for a type of mania. After the first week or two of the attack the temperature shows a tendency to become subnormal, and thereafter throughout the attack, and even after apparent recovery the temperature runs at about one degree below the normal, and at times the difference between the morning and evening temperatures may be so marked as to give the impression that the patient is passing through a mild septicæmic attack. Another peculiarity of the temperature is that it may be paradoxical, *i.e.* higher in the morning than in the evening. The paradoxical temperature is often associated with a paradoxical pulse rate. The paradoxical temperature and pulse are not however confined to patients suffering from "continuous mania."

In the early days of the attack the alimentary tract is much disordered. The teeth and lips become covered with sordes and the tongue is furred and foul. There is little desire for food, but thirst is generally present. I examined the stomach contents in two cases of acute mania at the onset of the disease and in both the digestive power was almost nil. At a later period of the disease, although still maniacal, I found the digestive power of the gastric juice of these same two patients very active. These observations are quite in keeping with what one observes in the appetite of these patients. At the onset of the

disease it is with great difficulty that the nurse can induce her patient to take a sufficient quantity of milk, and injudicious feeding often produces vomiting. In a fortnight or three weeks the same patient will eat ravenously and digest almost anything.

The circulatory system is but little disturbed. The pulse at the onset may be rapid and a little irregular. It rarely exceeds 100 beats per minute. It is not nearly so fast as the pulse of acute melancholia for instance. When a patient has suffered from a long attack of mania there is a tendency to heart weakness and failure.

The rate of breathing is slightly increased in bouts of excitement. Beyond a tendency to slight attacks of lobular pneumonia, which give rise to little local or general disturbance and are thus frequently missed, the respiratory system presents no peculiarities.

The skin is generally dry, but the palms of the hands and the soles of the feet may perspire freely. The hair is dry and brittle. Evanescent rashes and diffuse pustular eruptions are not uncommon.

The urine is scanty and high coloured at the onset of the attack, but I never detected any diminution of excretion of urea. Later in the disease the secretion of urine is abundant.

Erotic conduct and speech is common in both men and women. The menstrual function is never suppressed in women as it is in some other forms of mental disease.

There is general loss of sensibility to heat and pain, but the sense of touch is acute. The pupils are widely dilated, but react to light and accommodation and also to emotional conditions—for instance in a fit of anger the pupils may contract strongly. All the special senses except taste are acute,—according to Macpherson hyper-acute. The sense of taste is frequently disordered. Unless the patient is very ill or very badly nursed the organic reflexes of micturition and defæcation are kept under will power. The skin reflexes are slightly increased. The tendon reflexes, so far as I have been able to examine them, are not increased.

There is no paralysis or weakness of the voluntary muscles. The inco-ordination of movement, most strikingly exhibited by the facial muscles is of central origin.

The mental symptoms are—loss of self-control, characterised

by mental excitement and motor restlessness, loss of the power of attention, incoherence of speech and sleeplessness. Hallucinations are rarely present, but may be closely simulated by illusions of the hyper-acute senses. (Macpherson.)

The muscles, especially of the face and upper limbs, may show fine fibrillary tremors.

Probably the most interesting observations, as throwing light on the etiology of the disease, are those which have been made by my colleague Dr Peebles and myself upon the leucocytosis, which is present in every case of "acute continuous mania" which we have examined. We are not by any means the first workers in this field. Macphail, Krypsiakiewicz, Kroumbmiller, and others have examined the leucocytes in mania and other mental conditions. I am bound to admit that our observations do not bear out all the results of these earlier workers. I can only explain this discrepancy by the fact that these observers were misled by making isolated observations. We, on the other hand, made continuous observations on each patient for weeks and months. Further, we have not confined our observations to one type of case, but have examined every acute case admitted to the asylum, so that we have been able to compare our results in different cases. We have also made frequent control examinations on healthy subjects. In recording these observations, I start on the hypothesis that a leucocytosis between 6000 and 10,000 per c.mm. of blood is what one might expect to find in health. The blood was taken from the lobe of the ear; as nearly as possible at the same hour every day just before the midday meal. The numerical counts were made with Thoma Zeiss' hæmocytometer, according to the method of Coles', and never less than 30 fields were counted at each examination. The differential counts were made from films stained by various methods, and never less than 200 leucocytes were counted to obtain the various percentages.

The following were the results obtained. If the case was observed early in the acute stage in every case there was a leucocytosis of from 18,000 to 20,000 per c.mm., and sometimes the leucocytosis was even higher. The percentage of the polymorphonuclear cells was never below 70 per cent. This state of affairs did not last for many days, and then it was noticed that the leucocytosis fell sometimes as low as 10,000

per c.mm., but never lower. More generally the leucocytosis was 14,000 or 15,000 per c.mm. Along with this change the polymorphonuclear cells fell to 60 per cent., or even lower, but they never in any case came near 70 per cent. There was a corresponding rise in lymphocytes. This stage in most of the cases lasted for weeks, in others for months. The leucocytosis varied a good deal, corresponding to exacerbations of the disease. A slight increase of excitement was accompanied by a rise in the leucocytosis to perhaps 17,000 or 18,000, but the percentage of polymorphonuclear cells rarely rose above 60 per cent. A further change noticed during this period was the occurrence of eosinophiles, which were sometimes so numerous as to constitute a mild eosinophilia of three to five per cent. An eosinophilia did not, however, occur in every case. Whenever distinct mental improvement set in the leucocytosis again rose, and along with this rise the percentage of polymorphonuclear cells rose, sometimes above 80 per cent. always above 70 per cent. As recovery became complete the percentage of polymorphonuclear cells gradually fell until they averaged somewhere between 60 and 70 per cent., but the most interesting thing of all was the fact that the leucocytosis persisted for weeks and months after complete recovery. I have not yet had a single exception to this rule. It is highly probable that this leucocytosis persists indefinitely, and that it is a protective leucocytosis. If these observations are correct they prove that acute continuous mania is an acute infective disease, and that when recovery takes place a condition of immunity is established.

The persistent leucocytosis would indicate that although there is apparent recovery, the cause of the disease is still present in the body of the patient, and is only kept in check by the high leucocytosis maintaining an efficient immunity.

I have to thank my colleague, Dr Peebles, for his willing assistance in making these observations, and my friend Dr Lovall Gulland for much valuable advice with regard to stains and technique.

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## Abstracts

### ANATOMY.

#### **DIE VARIATIONEN IN DER LAGEUNG DER PYRAMIDEN-**

(92) **BAHNEN.** Prof. H. OBERSTEINER, *Arb. aus dem neurolog. Institute in Wien*, H. ix. 1902, S. 417.

PROF. H. OBERSTEINER in his paper analyses recorded cases of abnormal medullary pyramids, describes a similar specimen which he obtained from a dissecting-room body, and finally from these cases and from the condition of the pyramidal fibres in many vertebrates advances certain conclusions to account for these variations.

Two cases have been published by Pick, one by Van Gehuchten, and one by Kolk, in which the left pyramid was normal, but on the right side there was present an aberrant bundle, which passed down outside the olive. In Van Gehuchten's case, this bundle undoubtedly belonged to the pyramid, as a focal cerebral lesion was present with a resulting descending degeneration in this bundle as well as in the pyramid proper.

Prof. Obersteiner's own case differed from Pick's and Van Gehuchten's in that there was no or only slight asymmetry, and an abnormal condition was present on both sides.

The pyramidal fibres in the pons were normal. Both olives, however, were absent, the pyramids being broad and flattened and bounded laterally by a deep sulcus which separated them from the restiform body. The roots of the hypoglossal nerve emerged through the pyramids and by these, by external arcuate fibres and strong septa of neuroglia the whole area of the pyramids on transverse section was broken up into separate, oval, bundles.

The right side corresponded with Pick's case, a separate bundle, similar in size and position, and separated from the main pyramid by a slight furrow being present.

Many other cases have been noted, he adds, of aberrant pyramidal fibres not occupying the position of Pick's bundle.

Prof. Obersteiner then discusses the meaning of such variability in the pyramidal fibres, and is inclined to believe that the tendency to the presence of aberrant bundles is by no means rare. Not only so, but this is what we should expect to find.

He points out that in mammals there is a striking variability in the disposition of the pyramidal fibres. In most cases certainly

the crossed pyramidal tract is in the lateral column of the cord. In the rat and mouse among others, the crossed pyramidal tract lies alongside the grey commissure. In the sheep the fibres lie free in Burdach's column, and in the elephant they lie along the anterior fissure. Adding to this marked variability in the position of the pyramidal fibres in mammals, the occurrence of aberrant bundles in man, he is forced to the conclusion that it is only in the case of the pyramidal fibres that abnormality of position occurs. Not only in man but in vertebrates all the other tracts have a constant position. Flechsig, Prof. Obersteiner points out, associates this variability in the pyramidal decussation and position of the pyramidal fibres with their development.

Prof. Obersteiner agrees with Flechsig that the chief factor in accounting for the position which these fibres will take up is the late development of the pyramidal tracts. The pyramidal fibres are absent at the fifth month, receive their medullary sheath very late in foetal life, and at the time of their appearance find the other tracts already in position and have as it were to search for a course along which they can pass.

His general conclusions are (1) that variations in course and position occur in no tract so frequently and to so great an extent as in the pyramidal. (2) That this tendency to variation is explained by the fact that the pyramidal fibres belong to the tracts which are ontogenetically and phylogenetically the youngest.

The article is illustrated by five photographs.

EDWIN MATTHEW.

**ZUM STUDIUM DER MIT DEM THALAMUS OPTICUS UND  
(93) NUCLEUS LENTICULARIS IN ZUSAMMENHANG STE-  
HENDEN FASERZÜGE. VON JOHANN TARASEWITSCH, Arb.  
aus dem neurolog. Institute in Wien, H. ix., 1902, S. 251.**

THE author describes the lesion and consequent degenerations in a case of old hemiplegia, the patient having died as a result of a hæmorrhage in the thalamic region of the opposite side. No clinical notes are given. Softening and subsequent cicatrization had occurred in the following regions on the right side:—

1. The hindmost dorsal part of the pulvinar; the proximal region of the pulvinar near the posterior commissure.
2. The ventral, median and lateral nuclei, the "centre médian" and the nucleus arcuatus of the posterior third of the optic thalamus.
3. The dorsal part of the anterior third of the thalamus.
4. The hinder part of the internal capsule.

5. The putamen, part of the globus pallidus, the claustrum and the external capsule.

The remaining parts of the internal capsule and optic thalamus though not involved in the cicatrix were much atrophied.

The degenerations from this lesion were traced out by the Weigert method. The degenerated pyramid was much shrunken throughout, and was chiefly remarkable for its behaviour in the first cervical segment. Here, after the lateral pyramidal fibres had passed over to the opposite side, almost the whole of the uncrossed remainder passed outwards to occupy a ventro-lateral position in the region of Helweg's Dreikahntenbahn, thus forming a ventro-lateral pyramidal tract; no direct ventral pyramidal tract was present.

No definite change could be made out in the fillet, the central tegmental tract (centrale Haubenbahn) or dorsal longitudinal bundle. The most interesting degenerations were those in the thalamic and adjacent regions. The right fornix was atrophic from the corpus mammillare to the fimbria, the commissura supra-mammillaris also being almost completely atrophic; no definite atrophy was present in the cornu ammonis. The bundle of Vicq d'Azyr, involved at one part in the lesion was atrophic, as was the mesial part of the mesial ganglion of the corpus mammillare. The lateral nucleus of the corp. mam. was also atrophic, and the pedunculus corp. mam. was so small as to be hardly traceable, but the ganglion tegmenti dorsale (in which it ends) was much atrophied. The "Haubenbündel" was also affected at the point where it branches off from the bundle of Vicq d'Azyr. The author discusses the question of a direct communication between the fornix fibres and those of the bundle of Vicq d'Azyr, and concludes as opposed to Edinger and Wallenberg, that such a connection cannot yet be negatived with certainty, as in this case the fornix though not involved in the lesion was yet atrophic. The fasciculus retroflexus was very atrophic, and could be followed to the ganglion interpedunculare. The ganglion habenulae was partly involved in the scar tissue, and it was possible to divide it into three regions, i.e. (1) Dorsal—the region of the taenia thalami; (2) Ventro-lateral—the region of the fasciculus retroflexus, and (3) ventro-medial region of the peduncle. The superior cerebellar peduncle was also much atrophied, as a result, in the author's opinion, of destruction of the red nucleus and not of the thalamus. Atrophy of parts of the cortex cerebri was present, but on account of the size of the lesion, it was impossible to determine whether this atrophy was as a result of destruction of the internal capsule, lenticular nucleus or optic thalamus.

STANLEY BARNES.

- EIN VERGLEICHEND - ANATOMISCHER BEITRAG ZUR**  
 (94) **KENNTNIS DER HAUBENFASERUNG UND ZUR FRAGE**  
**DES CENTRALEN TRIGEMINUS-VERLAUFES.** Von R.  
 HATSCHKE, *Arb. aus dem neurolog. Institute in Wien*, H. ix.,  
 1902, S. 279.

IN this paper the author draws attention to the presence in certain mammals of a tract which closely corresponds in position to that first described by Wallenberg as the secondary trigeminal tract from experiments on the rabbit, and later from a pathological specimen in man. It is considerably larger in the ungulata (horse) than in other mammals, and consequently more easily followed.

The fibres take origin in the nucleus of the sensory V; the majority decussate in the dorsal part of the raphé, and then form a bundle ventromedian to the superior cerebellar peduncle. Further cerebralwards it is found among the decussating fibres of the peduncle, and in the midbrain lies dorsolateral to the dorsal longitudinal bundle on the border of the central grey matter. It is later pierced by the fibres of the posterior commissure, and in the thalamus by the fasciculus retroflexus. Its ventral fibres end in the ventral nucleus of the thalamus, the dorsal in a cell mass which probably corresponds to the centre médian and lateral nucleus.

The paper forms a valuable confirmation of Wallenberg's work on this important tract.

GORDON HOLMES.

- BEITRAG ZUR ANATOMIE DER RIECHSTRAHLUNG VON**  
 (95) **DASYPUS VILLOSUS.** Von E. ZUCKERKANDL, *Arb. aus dem*  
*neurolog. Institute in Wien*, H. ix., 1902, S. 300.

THIS paper deals chiefly with the cingulum and those strands of fibres which lie in the gyrus supra-callosus (striae Lancisii) as they are present in the armadillo, where the olfactory apparatus is strongly developed.

The chief conclusions are: The cingulum arises in the gyrus fornicatus and the convolution which lies dorsal to it. It runs forwards on the surface of the corpus callosum, lateral to the supra-callosal gyrus, but gradually diminishing in size owing to some of its fibres passing ventralwards through the corpus callosum. At the oral end of the latter its remaining fibres turn directly to end in the olfactory lobes. The fibres which pass through the corpus callosum either go to form the fornix longus, or running forwards on its ventral surface also enter the olfactory lobes.

The fibres of the supra-callosal gyrus (cingulum gyri supra-callosi) are arranged in three layers, and take origin in the cornu Ammonis and callosal convolution. Practically none of these pierce the corpus callosum, but at its frontal end either join the septal bundles to end in the olfactory lobes, or split up in the median cortex of the hemisphere. In this way the gyrus fornicatus as well as gyrus supra-callosus is connected with the olfactory lobes.

The stria terminalis which passes forwards from the gyrus hippocampus, divides above the commissura anterior into a precommissural and a retrocommissural bundle, each of which ends in the basal cortex, though some fibres of the former probably decussate in the commissure.

GORDON HOLMES.

**ÜBER EIN AUSTRALIERGEHIRN, NEBST BEMERKUNGEN**  
(96) **ÜBER EINIGE NEGERGEHIRNE.** Von Privatdocent Dr  
J. P. KARPLUS, *Arb. a. dem neurolog. Institute in Wien*, herausgegeben von Prof Dr Heinrich Obersteiner, H. ix., 1902, S. 118.

THIS description is welcome, since it is the first account of the native Australian brain to appear in literature, and consists in a full account of the brain of one of those natives known as the "Barron Fall Boys," who died in Brisbane hospital, and whose brain was sent to the Vienna University Neurological Institute. The author first points out that the racial differences once supposed to be so marked in the brains of lower races are no longer to be believed, and that a much more extended series of observations must be made before definite statements can be formed as to the inferiority in weight or in complexity of convolutions of brains from lower races of mankind.

The brain, with fine membranes, in this case weighed 1382 g. approximately—since the back part of the pons and the medulla were wanting—and, subtracting 1 per cent. since it had been hardened in 10 per cent. formal solution, the weight when fresh was probably 1368 g. As a whole, the brain appeared to be moderately well convoluted, of a good size, and in its configuration showed no special features beyond a peculiarity of the right occipital convolutions on the outer surface.

In this region there was an increase of the front part of the occipital lobe on the outer surface, so that its anterior end formed a sort of operculum which projected forward, and covered over the end of the bridging convolutions connecting the occipital and parietal lobes.

The author gives a summary of the views held as to the homology in the human brain, of the "affenspalte" which is frequently

supposed to be represented by the bifid end of the ramus occipitalis of the intra parietal sulcus.

The remainder of the work consists in a brief description of the characters of three negro brains, which are mainly negative, as far as racial characters are concerned.

DAVID WATERSTON.

### PHYSIOLOGY.

**THE SUBMAXIMAL ELECTRICAL RESPONSE OF NERVE TO**  
(97) **A SINGLE STIMULUS.** FRANCIS GOTCH, *Journ. Physiol.*,  
Dec. 1902, p. 395.

THE author first of all points out how the extent of the mechanical response of muscle to a submaximal stimulus can be recorded with sufficient accuracy by the isometric method, or isotonicity by the method of "arrest." The muscle twitch, however, cannot be regarded as a true indication of the extent of the excitatory change in nerve, and the only physical indication that can be made use of in the nerve itself is the electrical disturbance which accompanies the passage of a nerve impulse. Owing to the great variations which take place in the excitability of nerve from changes of temperature and as a result of stimulation, great precautions have to be taken to maintain the unvarying character of all conditions other than the intensity of the exciting stimulus. To ensure this the nerve to be examined was placed in a special moist chamber which stood in an ice bath, the whole being enclosed in a large ice safe. A thermoelectric junction near the nerve balanced against a similar junction kept in a small ice calorimeter at 0° C., and connected to a low tension galvanometer at once indicated any change of temperature in the moist chamber. The sciatic nerve of *Rana temporaria* was carefully prepared, and soaked for eighteen hours in physiological saline to allow excitatory changes due to injury to subside. It was then fixed in the moist chamber by mimosa spines, and non-polarisable electrodes were placed on the nerve, one near the knee and the other 20 cm. higher up. The electrodes were connected to a capillary electrometer, the meniscus of which could be photographed on a plate fixed to a pendulum. The exciting current was obtained by opening the primary circuit of an induction coil without central core, and one Daniell cell, the stimulus being passed through the spinal end of the nerve by a pair of steel needles placed one on each side of the whole trunk. By means of a special electro-magnetic key operated on by the swing of the pendulum the opening of the exciting circuit was of a constant velocity, and the resulting induced current constant

for any given position of the secondary coil. The moment of stimulation, whether maximal or submaximal, was also recorded on the plate. After everything had been fitted the preparation was allowed to rest for a time, then the experiment began by observing the positions of the secondary coil necessary to produce a minimal and maximal response as recorded by the muscle twitch. After each observation the nerve was allowed to rest, and the temperature of the moist chamber kept constant. A number of observations were made and the curves analysed so as to give curves of E. M. F. production. The E. M. F. curves of maximal and submaximal response were placed on the same sheet for comparison. In the uninjured nerves the curves were diphasic, and the first phase in both maximal and submaximal response began '0014" after application of the stimulus. As the point of stimulation remained unaltered throughout the experiment it followed that the rate of conduction of maximal and submaximal impulses was the same, its speed being 14 metres per sec. at the temperature of 35° C. The highest potential in either case coincided in point of time, and was reached '0024" after excitation. The subsidence of the first phase occurred in both '0045" after excitation. The second phase began in both '0045" after excitation and ended '011" later.

The conclusions from these experiments are that the electrical response of a nerve to a single stimulus varies as to its magnitude in correspondence with variations in the exciting efficiency of the stimulus, but that the time relations are practically identical whatever the magnitude of the response.

A second series of experiments were made to ascertain how far the submaximal effects were due to excitation of only a certain proportion of the nerve fibres in the whole nerve trunk. This was done by excitation of the individual branches that unite to form the sciatic nerve in the frog; arrangements were also made so that the whole trunk could be stimulated, and a comparison of effects thus obtained. Stimulation of one branch even when fully adequate to arouse the excited tissue to its utmost was found to give in the nerve trunk containing other nerve fibres a submaximal electrical response.

The records show that the existence of maximal excitatory states in a limited number of nerve fibres in a nerve trunk give rise to submaximal effects, the stimulus spreads by virtue of its increased intensity so as to arouse more nerve fibres; the records also suggest that when all the elements are involved the response becomes maximal.

Similar experiments were performed on voluntary muscle in which the fibres are physiologically distinct. Stimulation of a branch of the sciatic plexus by section caused a submaximal twitch, section of the nerve trunk produced a maximal effect.

The author concludes that the number of elements excited is the potent factor in producing the magnitude of response, when all are excited the response is maximal. Where the elements are in physiological continuity, as in the case of the muscle fibres of the heart, there is no distinction between responses and no differentiation of maximal from submaximal stimuli.

PERCY T. HERRING.

**QUELQUES NOUVELLES DONNÉES SUR LA PHYSIOLOGIE DES**  
(98) **RÉFLEXES TENDINEUX.** STCHERBAK, *Rev. Neurolog.*, Jan.  
15, 1903, No. 1, p. 17.

THESE experiments were performed on rabbits. A large tuning fork (33 per sec.), driven by an electro-magnet, was fixed to a strong metal support, against the edge of which the limb of the animal was firmly pressed, so that the vibrations of the tuning-fork were communicated to the knee joint on one side. As the result of the local application of this mechanical irritation, the excitability of the reflex spinal arc was greatly increased. The knee-jerk on the side stimulated was exaggerated; knee clonus was easily elicited, and sometimes appeared spontaneously, and in some cases unilateral spontaneous spasmodic tremors were also observed. This increased excitability persisted long after the peripheral irritation had ceased. In one case, *e.g.*, where the stimulation had been continued for an hour, knee clonus was induced, by repeated passive movements, 24 days after the experiment. In general, the more powerful the mechanical irritation, and the longer the period of its application, the more easily could the spasmodic phenomena (knee clonus, etc.) be induced by passive movements, and the longer was the interval after the experiment within which they could be induced. This mechanical irritation would appear to be a specific stimulus for the deep endings of the sensory nerves which are brought into play in the patellar reflex.

On dividing the spinal cord in the mid-dorsal region, *i.e.* above the patellar reflex arc, under the influence of the local vibrations, there was a permanent unilateral exaggeration of the knee-jerk, but neither knee clonus nor spasmodic tremors could be called forth.

In another series of experiments the vibrations were communicated to the vertebral column in the lower dorsal and other regions. This was followed by spasmodic phenomena (knee clonus, etc.) in both hind limbs, and the "charge" remained latent for several days after the experiment. The muscle tonus was never increased; sometimes, in fact, it was diminished.

The author concludes that there is a complete analogy between this phenomenon in the lower sensori-motor somatic centres, and



the phenomenon of memory in the higher psychical centres. In the lower centres the mechanical vibrations communicated to the deep terminations of the sensory nerves, are stored up, so to speak, and remain latent until called forth again in the form of clonic or spasmodic movements. In a similar way, sensory impressions from the eye or ear, say, are stored up in the psychical centres, and after long intervals, are capable of being called forth again, not as visible movements, but as subjective sensations. In other words the spinal cord has the power of reproducing or "remembering" mechanical vibrations, just as the telencephalon has the power of reproducing or remembering past sensory impressions.

SUTHERLAND SIMPSON.

**THE NATURE OF THE LESIONS WHICH HINDER THE  
(99) DEVELOPMENT OF NERVE CELLS, AND THEIR PRO-  
CESSES.** H. K. ANDERSON, *Journ. Physiol.*, Dec. 1902, p. 499.

THIS paper deals with the effects produced in nerve cells and fibres by the section of nerves in very young animals.

The right sciatic nerve was divided in a kitten 11 days old, and the animal killed after 44 days had elapsed. The 6th and 7th lumbar, and the 1st sacral ganglia were found to be smaller on the right side than on the left; the majority of the nerve cells in these ganglia on the right side were smaller, and their granules less deeply staining than those of the ganglia on the left side. The posterior roots of the smaller ganglia were less well developed, and their individual fibres smaller than those of the corresponding ganglia on the opposite side. In the spinal cord, by Weigert's method, an area of smaller fibres was found in the posterior columns of the right side. Division of the peripheral fibres of a spinal ganglion therefore hinders the development of the cells of that ganglion, the fibres of its posterior root, and of its fibres in the posterior columns of the spinal cord. There were also fewer cells to be found in Clarke's column on the right side than on the left in the 2nd and 3rd lumbar segments.

The next experiments were to determine the effect of section of posterior roots on the development of the spinal ganglia and their peripheral processes. Young rabbits and kittens were used, and killed from 26 to 52 days after section of several posterior nerve roots. In every case the ganglia of the injured side were as well developed as the corresponding ganglia of the opposite side; the cells were as large, and as numerous. No change was found in the number and size of the peripheral fibres of these ganglia. Injury, therefore, of the posterior nerve roots does not

check the development of the ganglia, nor of their peripheral processes.

Section of the vagus nerve above the ganglion of the trunk in a young rabbit caused no change in the cells of the ganglion, nor in their peripheral processes. Section of the vagus in another animal, low down in the neck, produced much atrophy of the cells of the ganglion.

Section of posterior nerve roots did not, in one case at least, produce any atrophy of the cells in the anterior horn of the spinal cord. In all cases where posterior nerve roots had been cut the part still attached to the ganglion showed considerable atrophy, although the nerve cells and their peripheral processes were unaffected.

The remainder of the paper deals with the sympathetic nervous system. Section of the præganglionic fibres was found to greatly hinder the development of the central end of the nerve. The fibres in the central end of the divided cervical sympathetic nerve were, 39 days after the lesion, smaller and far fewer than those of the uninjured nerve.

Section of postganglionic fibres, the branches of the superior cervical ganglion being taken, caused in very young animals a marked diminution in the size and number of the præganglionic fibres. The cells in the ganglion were fewer and atrophied. There was no increase of connective tissue in the ganglion. Control experiments showed that the changes in the præganglionic fibres were not brought about by alterations in the blood supply, or exposure and handling of the ganglion during operation.

Division of the præganglionic fibres produced no changes in the ganglia, nor in the postganglionic fibres. There was no evidence of "disuse atrophy."

The origin of the cervical sympathetic fibres in the spinal cord was found to be in the small cells of the lateral horn of the same side, these cells alone were atrophied after division of the nerve.

The author then discusses the reasons for the arrest of development or the occurrence of atrophy after these lesions. He suggests that the lesion disturbs the "chemico-physical equilibrium of an anatomically continuous (neuro-muscular or neuro-epithelial) chain of cells by separating the non-nervous tissues from the nervous." Section of the posterior nerve roots checks development in those portions of them still attached to the spinal ganglia, because the chemico-physical equilibrium in these processes is maintained not only by the spinal ganglion cells, but also by intraspinal cells with which these processes are anatomically continuous.

PERCY T. HERRING.

**PSYCHOLOGY.**

**AN INVESTIGATION OF FECHNER'S COLOURS.** FLORENCE  
(100) BAGLEY, *Am. Journ. Psychol.*, Oct. 1902, pp. 488-525.

If a disc composed of black and white sectors is rotated with moderate rapidity, colours appear upon the anterior and posterior edges of the sectors. The explanation of these colours is obscure, owing to our ignorance of the exact processes which take place in the retina during and after stimulation. Experiments were made in the Cornell Laboratory with a series of 97 top discs, fully described in the paper, and one called the Helmholtz disc. These were rotated by means of a Crocker-Wheeler motor, whose speed was reduced by a Pillsburg speed-reducer. The room was darkened and the discs were illuminated by a Welsbach gas-burner.

As a result of the experiments it is concluded that colours produced by the rapid alternation of black and white sectors, are further dependent upon the duration of stimulation, and the co-excitation of black and white. They are also dependent in less degree upon length and width of lines; upon their position within the sector; and, as are all other subjective states, upon bodily conditions, practice, fatigue and attention. These conditions are necessary for the production of the colour itself; but after it is once present its quality may be changed by change in rotation of the disc, change in amount of light, and by addition of background colour. The results of the experiments are regarded as opposed to the Helmholtz theory of visual sensation, and the author finally selects Ebbinghaus's modification of Hering's theory as that which furnishes the most adequate, the most detailed and the most concrete explanation of the experimental results.

W. B. DRUMMOND.

**ZUR PATHOLOGIE DES BEKANNTHEITSGEFÜHLS (BEKANNT-  
(101) HEITSQUALITÄT).** A. PICK, *Neurol. Centralbl.*, Jan., 1903,  
S. 2.

THE object of this paper is to direct attention to certain factors already recognised in normal psychology, which may assist in the understanding of some cases of morbid psychology. These factors have already been named, the feeling of familiarity or the certainty of recollection. Volkelt has already drawn attention to the fact that the distinctness of a recollection is of less importance in its recognition than the feeling of familiarity associated with it, and that this certainty of recollection qualifies all individual past experiences. In certain cases of hysteria and of epilepsy a careful

examination of the patient shows that without any psycho-sensory disturbance he has a feeling of being out of touch with his surroundings due to the loss of this sense of familiarity. This may also lead to feelings of fear, paramnesia, or double consciousness. Its converse may also lead to a feeling of familiarity with a situation undoubtedly experienced for the first time. Similar conditions may be seen in general paralysis, confusional insanity and paranoia, and no doubt in many cases of mental disease of various kinds.

JAS. MIDDLEMASS.

### PATHOLOGY.

**ZUR FRAGE VON DER AUTOGENEN NERVENREGENERATION.**  
(102) TION. ALBRECHT BETHE, *Neurolog. Central.*, Jan. 16, 1903, S. 60.

THIS is Bethe's reply to Münzer's paper published in the 1st December number of the *Neurologisches Centralblatt*. He points out that in his own experiments a portion of sciatic nerve was excised in such a way that no axis cylinders could possibly reach the peripheral end of the nerve from the central, and that the peripheral end of the divided nerve was not connected with any neighbouring undivided nerve by anastomosis. After regeneration had occurred in the peripheral unconnected end Bethe got contraction of the muscles by stimulating the peripheral end with a weak current; this could not be due to direct stimulation of the muscles, and it did not occur when the central end was stimulated or when neighbouring undivided nerves were stimulated. Bethe obtained the same results even when the posterior nerve root ganglia and motor roots were removed.

Bethe takes exception to Münzer's use of rabbits, which the latter exclusively employed, and points out that with dogs he has had fewer failures, while he admits that in a small percentage of experimental cases regeneration does not occur. Bethe also suggests that as his mode of operating has not been described, Münzer may not have carried out his experiments satisfactorily. He concludes by denying that a mistaken interpretation of his microscopic sections is a possible explanation of the results he has obtained.

ROBERT A. FLEMING.

**ZUR FRAGE VON DER AUTOGENEN NERVENREGENERATION.**  
(103) EGMONT MÜNZER, *Neurol. Centralbl.*, Jan. 16, 1903, S. 62.

THIS paper is a reply to the preceding criticism of Bethe's. Münzer points out that he has followed Bethe's methods of opera-

tion, and that in neither the matter of the age of the animals used for experimental nerve section, nor the mode in which these operations were carried out, is adverse criticism warranted. Münzer denies that after 150 days he found no myelinated nerve fibres in the peripheral end of the divided sciatic, and refers to his paper in which he describes a few myelinated fibres among the fibres of the calf muscles.

He does not consider the muscular contraction obtained by electrical stimulation of the peripheral end of the divided nerve is an argument in favour of Bethe's contention, and antagonistic to his own position. Münzer expresses his regret that Bethe has not referred to the young nerve fibres which he found at the site of section, and which he says he believed might be connected with the central end of the nerve, although he left the matter an open question to a considerable extent.

ROBERT A. FLEMING.

**NÉVRITE RADICULAIRE SUBAIGÜE. DÉGÉNÉRESCENCES (104) CONSÉCUTIVES DANS LA MOELLE (RACINES POSTÉRIEURES) ET DANS LES NERFS PÉRIPHÉRIQUES (RACINES ANTÉRIEURES).** J. NAGEOTTE, *Rev. Neurolog.*, 1903, p. 1.

THIS paper deals with the changes in cord and peripheral nerves following a sub-acute inflammation of the lumbo-sacral nerve roots. The patient was a woman, aged 28, who died with the symptoms of a moderately acute transverse myelitis. There was paraplegia and anaesthesia of the lower limbs, with atrophy and loss of tone in the muscles. The sphincters were also involved. On post-mortem examination there was found (a) a circumscribed lesion on the inferior surface of each frontal lobe; (b) a focus of transverse myelitis, 2 cm. in length, at the level of the tenth dorsal segment; and (c) an inflammatory condition of the lumbo-sacral nerve roots, which had, however, no connection with the area of myelitis. Above the myelitis was an ascending degeneration in the columns of Goll, in the direct cerebellar tracts and in Gowers' tracts; and below the lesion, a descending degeneration in the pyramidal tracts. In the posterior columns, commencing at the level of the third sacral nerve, a degeneration was to be seen on both sides, but more marked on the left. It increased in extent as it passed upwards, till at the level of the first sacral it occupied the greater part of the posterior columns. Upwards from this it varied somewhat in shape and ultimately was lost in the focus of myelitis. Immediately outside the cord the posterior roots presented marked degeneration. But on tracing these roots still further outwards, each successive section showed a diminution in the number of degenerate fibres, till

at the point where the posterior roots joined the anterior, degeneration had entirely disappeared. The degeneration in these sensory roots, then, was more marked the nearer they were to the cord, and more marked still after they entered the cord. In other words, the degeneration was most marked in the most peripheral portions of the neurone.

As to the anterior nerve roots, they were normal immediately on leaving the cord, but degeneration appeared as they left the sub-arachnoid space. (The histological changes in these anterior roots are described in detail.) Secondary degeneration was found in the peripheral motor nerves; but the sensory nerves also showed a fairly extensive degenerative change, particularly at their extreme periphery. This last, however, the author considers as due to the general cachexia from which the patient suffered.

The cells in the anterior horns corresponding to the degenerated anterior roots, were in a chromatolytic condition, and, indeed, presented the typical "réaction à distance" appearance. But, on the other hand, the most of the cells in the posterior root ganglia were normal.

W. K. HUNTER.

**PATHOGÉNIE DU TABES DORSAL.** J. NAGEOTTE, *Presse Méd.*, (105) Jan. 3, 1903.

In a preceding article\* M. Nageotte drew attention to the early appearance of a syphilitic meningo-myelitis in all cases of tabes, and to its direct action in producing a root neuritis, which is the cause of the degeneration of the nerve fibres in the posterior columns of the cord. In this paper he describes in detail the changes found in the inflammatory focus.

The root neuritis may invade the nerve root in all its length and in all its thickness.

In a transverse section it is possible to distinguish, (1) an inflammation of the intrafascicular tissue and of the very thin sheath which surrounds the fasciculus, which are derived from the pia mater—an endoneuritis: (2) an inflammation of the connective tissue derived from the arachnoid tissue—an epineuritis: this forms an inner layer to (3) a sheath derived from the dura mater, which is the first to be affected, giving rise to a perineuritis.

This perineuritis, which is characterised by an infiltration of fixed cells, lymphocytes and a few epithelial cells, usually attacks the posterior root in its middle-third, while the focus in the anterior root, which is also frequently affected, is situated a little nearer the cord. It does not spread downwards to the ganglion.

\* *La Presse Médicale*, 10th December 1902.

With it the vessels are often involved, and amyloid bodies are often seen lying around them in concentric layers.

The inflammatory process tends to spread inwards to the fascicular sheath and the intrafascicular connective tissue which are derived from the pia mater. This fascicular sheath seems for a time to act as a barrier to the spread of the inflammatory process, and it is only after it has given way that the degeneration in the nerve fibres begins. In very early cases a local atrophy of the fibres may be seen at the level of the inflammatory lesion, which is sufficient to injure the distal portion of the nerve and to cause the slow atrophy which is characteristic of the disease, but is not sufficient to destroy the fibre by a true wallerian degeneration. A similar condition is frequently met with in cases of cerebral tumour.

This atrophy usually commences in the parts of the nerve which are farthest removed from the trophic ganglion, and spreads towards the centre; and the author states that even in wallerian degeneration, after section of the nerve, the first changes are seen at the periphery and spread towards the point of section. In the anterior roots the degeneration extends towards the cord for a short distance, and is soon followed by a regeneration of the nerve fibres. This regeneration is not seen in the central stump of the posterior root, probably because the spread of the endoneuritis downwards into the ganglion destroys this portion of the fibre. In older cases the chronic inflammation leads to the formation of a cicatrix, which often has an atrophic appearance; but sometimes there is an enormous hypertrophy of fibres and cells, and in these latter cases cavities and hæmorrhages are often found in the hypertrophied tissues, probably as a result of hyaline changes in the vessels. In even advanced cases small masses of active round cells are generally present, and these are the cause of the progressive character of the disease.

In discussing the question of the cause of the variety of lesions which are seen in different cases of tabes, the author draws attention to two very important factors which assist in determining the seat and the extent of the affection, viz., the virulence of the infection, and the individual resistance of the tissues, natural or acquired. Both of these factors will vary in different cases, but he has found that in the same case, in which, of course, all the tissues are exposed to the same infection, the parenchymatous changes in the different roots, and in the different fasciculi of the same root, vary with the degree of the interstitial lesion.

He expresses the conclusion that tabes is the result of a syphilitic transverse interstitial neuritis of the nerve roots which is derived from a preceding chronic meningo-myelitis.

As regards treatment he recommends that lumbo-sacral puncture

should be performed in all cases in which there is the slightest suspicion, so that advantage may be taken of the cytological examination in forming a diagnosis, and specific treatment may be started even before there are any signs to prove that the posterior roots are affected.

R. G. Rows.

**LES LÉSIONS DU SYSTÈME LYMPHATIQUE POSTÉRIEUR DE  
(106) LA MOELLE SONT L'ORIGINE DU PROCESSUS ANATOMO-  
PATHOLOGIQUE DU TABES.** Par PIERRE MARIE et GEORGES  
GUILLAIN, *Rev. Neurol.*, Jan. 31, 1903, p. 49.

THIS paper brings into prominence a factor which has been practically ignored by previous investigators into the pathogeny of tabes. The authors draw attention to the well-known fact that the leptomeninges covering the posterior columns of the cord in the dorsal and lumbo-sacral regions are opaque and thickened. They regard this change as the main cause of the lesions of the cord in tabes. They contend that the primary change in the cord is not really due to a root lesion, but only apparently so, and that the pseudo-systematisation, as they term it, is due to the arrangement of the lymphatic channels in the posterior columns, and to the septa which pass inwards from the pia mater. That the degeneration does not correspond to the distribution of any root or groups of roots is shown by the examination with the aid of Marchi's method of cases of recent tabes. The black droplets which indicate degeneration of the nerve fibres are found scattered irregularly in the dilated lymph spaces throughout the posterior column, some even reaching the ependyma. In severe cases where the limits of the posterior lymphatic system have been exceeded, the portion of the lateral column adjacent to the posterior cornua may also contain black droplets. Such lesions as are found in the root ganglia are quite insignificant and in no way competent to explain the alterations in the cord. Nor are any of the other recent explanations such as are adduced by Nageotte, Thomas and Hauser in any way satisfactory.

The limitation of the meningitis to the posterior columns is attributed to the distribution of their lymphatic system. Between the layers of the pia mater there is a special system of lymph vessels, which has practically no communication with those of the pia of the lateral columns. Within the posterior column itself there are no true lymph vessels, but merely perivascular sheaths and spaces in which the lymph circulates. Injections into the membranes of coloured liquids—made in small quantity and with precautions to avoid laceration of the tissues—by d'Abundo and



Guillain, and of microbes by Homen, enter the posterior columns and ascend towards the central canal. From these and other facts the authors infer that there is a special lymphatic system for the posterior columns, the pia mater which covers them, and the posterior roots. They regard the initial lesion in tabes as a syphilitic affection of the posterior lymphatic system. If these observations are substantiated by future research, it is not too much to say that they will entirely revolutionise our opinions as regards the origin of tabes.

ALEXANDER BRUCE.

**DU PARASITE TROUVÉ DANS LE SANG DES ÉPILEPTIQUES.**

(107) M. BRA, *Rev. Neurolog.*, No. 10, 1902, p. 447.

THE author examined the blood of epileptics immediately before and during the attack, and also in the interval between seizures. The blood was taken from a vein after careful sterilization of the skin with alcohol and ether. The syringe used in the operation was boiled, and the needle passed through the flame. In fresh preparations of blood, a highly mobile parasite was detected, consisting of single small elements, or of worm-like structures composed of many elements, feebly refractile, and exhibiting serpentine movements.

In coloured specimens the particles composing the chains were somewhat unequal in size, but the most typical shewed a small chain terminating at each end with a rounded particle larger than the rest. The number of chains varies in different individuals, being abundant in some and completely absent in others. By taking the blood at favourable periods, *e.g.* before an attack, during incomplete crises, or during an attack of giddiness, the author has been able to cultivate the organism from the blood in 70 out of 100 cases. When grown, the cultures were injected into rabbits and convulsions resulted.

The organism stained badly with Gram, but well with hydro-alcoholic and aqueous solutions of aniline dyes. Bra gives his organism the name of Neurocoque, and supposes that it lives at the expense of the blood elements, increases in quantity on the approach of fits, and diminishes or disappears after the convulsive seizures.

He concludes that the organisms are the pathogenic agents of epilepsy, that they induce a series of infections, and that the symptoms are the result of irritative action on the nervous system by these organisms circulating in the blood.

DAVID ORR.

**DU PARASITE TROUVÉ DANS LE SANG DES ÉPILEPTIQUES.**  
 (108) —**SON AGGLUTINATION PAR LE SÉRUM DES ANIMAUX**  
**INFECTÉS ET PAR LE SÉRUM DES ÉPILEPTIQUES.**  
 M. BRA, *Rev. Neurolog.*, No. 1, 1903, p. 19.

THE author has studied the action of the serum of animals infected with the parasite above described and the action of the serum of epileptics. Rabbits received injections of cultures in bouillon for three months, and the serum derived from them produced agglutination of the organism, causing the chains to become grouped together and immobile. Epileptic serum produced the same result.

He concludes that epilepsy produces the agglutinative power, and that the diagnosis of true epilepsy can be established upon—

- (1) The presence of parasites in the blood.
- (2) Their agglutination.
- (3) The serum reaction.

DAVID ORR.

**RICERCHE BATTERIOLOGICHE NEL SANGUE DEGLI EPI-**  
 (109) **LETTICI.** CARLO BESTA, *Riv. Sperment. di Freniatria*, f. 2-3,  
 1902, p. 309.

BESTA conducted a systematic research upon 22 epileptics before, during, after the fits, and in the period between attacks. After fits the blood was taken three or four times at hourly intervals, occasionally every half-hour. Before inserting a sterilized syringe into the vein of the arm, he thoroughly sterilized the skin with carbolic acid, absolute alcohol and sulphuric ether. Small quantities of blood were then taken and cultivated in broth for eight days.

Altogether 125 examinations were made, and 375 tubes inoculated, *i.e.* 3 tubes from every case. Only 5 cases yielded a positive result, and even then only in one of the 3 tubes inoculated.

In one of those cases the syringe was overturned; in two the patient was excited, and examination rendered difficult. Again in two cases only, were organisms found at the time of a fit. In all the 5 cases the organisms obtained were those commonly found in asylums and on the skin of patients, *e.g.* staphylococci, bacterium coli, micrococcus tetragenus, and in one case an unidentified spirillum.

The author therefore concludes: (1) that epilepsy and its phenomena cannot at present be referred to the action of a special micro-organism; (2) positive results may be attributed on the one hand to auto-infection, and on the other to pure defects of technique.

DAVID ORR.

**THE HISTOLOGICAL APPEARANCES OF THE NERVOUS  
(110) SYSTEM IN KRAIT AND COBRA POISONING. W. K.  
HUNTER, *Glasgow Med. Journ.*, Feb. 1903, p. 98.**

THIS paper is a sequel to one by Captain Lamb, of the Indian Medical Service, on "Snake Venoms: Their Physiological Action and Antidote," and its purpose is to describe the results of the microscopical examination of the nervous systems of animals killed by certain of these poisons. Two monkeys were killed with krait poison, and one monkey and three rats with cobra poison. The first monkey died paralysed on the sixth day after injection of krait venom, and the second monkey on the tenth day after injection. The monkey injected with cobra poison died paralysed in six hours. Speaking generally the changes in all three monkeys were much alike, for all showed an acute degenerative change, both in ganglion cells and nerve fibres. The appearances might be likened to those found in the most acute cases of acute insanity, for the changes in the two conditions are very much the same. In the three monkeys few normal ganglion cells were anywhere to be found, the chief changes being a disappearance of the Nissl bodies with, in some cases, vacuolation of the cell. The number of fibres affected in the central nervous system was considerably less than the number of degenerative cells, but the peripheral nerves, particularly in the cobra poisoning, showed a very marked degeneration.

In the three rats the changes were much the same as in the monkeys, but in rat 3, which had been kept alive for 45 hours by means of anti-venom serum, the degeneration and disintegration of the ganglion cells was most extreme.      **AUTHOR'S ABSTRACT.**

**CLINICAL NEUROLOGY.**

**PARALYSIS OF ALL FOUR LIMBS AND OF ONE SIDE OF THE  
(111) FACE WITH DISSOCIATION OF SENSATION, DEVELOP-  
ING IN A FEW HOURS AND RESULTING FROM  
MENINGO-MYELOENCEPHALITIS. CHARLES K. MILLS  
and WILLIAM G. SPILLER, *Journ. Nerv. and Ment. Dis.*, Jan.  
1903, p. 30.**

THE patient was a negro, aged 45 years. Syphilis two years before. Four months before illness, fell on left shoulder, followed by severe pain in left arm, recurring from time to time. Illness began with sudden palsy of left arm followed by palsy of left leg, right side of face, right arm and right leg, in that order during a space of six hours. No pain nor loss of consciousness accompanied the onset of paralysis, nor was he aphasic. Severe headache followed the onset of the illness.

## Summary of physical state :—

Fully conscious. No aphasia. Bilateral ptosis. Pupils equal and reacted well. Ocular movements normal. Right face paralysed. Complete flaccid paralysis of upper limbs (trapezius excepted) and of lower limbs. The deep reflexes were lost at first, afterwards they were feeble. Babinski's reflex was absent.

The anæsthesia and analgesia were absolute in the lower extremities and on the right side of the chest and abdomen, relative on the left side of the chest and abdomen and on the left upper limb and on the distal portions of the right upper limb.

Tactile anæsthesia was only present on the distal portion of the right leg. Respiration was entirely costal. Incontinence of urine appeared late in the illness, and death occurred from asthenia on the thirteenth day.

Anatomy. Throughout the brain and spinal cord intense round-celled infiltration was present beneath the pia mater and the walls of some of the pial vessels were thickened. The right facial nucleus was exceedingly degenerated, the left slightly so.

The fourth, fifth and sixth cervical segments were completely softened by inflammatory changes and collections of small bacilli were found within the spinal cord at the eighth cervical segment. In the region of the softening there was intense thickening of the blood-vessels.

JAMES COLLIER.

**ASTEREOGNOSIS IN TABES DORSALIS.** G. E. RENNIE, *Brit. (112) Med. Journ.*, Feb. 7, 1903, p. 297.

NOTES are given of two cases of tabes dorsalis in which astereognosis was present. In the first case, there was complete inability to recognise the nature of objects placed in the left hand: this hand showed loss of the spacing sense, the pressure sense, the sense of position and of weight, and there was diminished sensibility to touch, pain and temperature over the ulnar side of the left forearm and hand, and ataxia of the hand was present. In the second case, the patient was unable to tell the nature of objects placed against the sole of the foot or under the toes: the left foot showed blunting of tactile, painful and thermal sensations, also loss of the spacing sense and of the sense of the position of the foot and toes.

Some remarks are made on the special sensations concerned in the stereognostic sense, and the loss of this sense in tabes is attributed to a dissociation of these special sensations, which must take place "either in the spinal cord from defective conduction or in the peripheral sensory receptive mechanism."

ASHLEY W. MACKINTOSH.

**STUDIEN ÜBER SEGMENTALE SCHMERZGEFÜHLSSTÖRUNGEN**(113) **AN TABETISCHEN UND EPILEPTISCHEN.** Von Dr L. J. J.**MUSKENS**, im Haag *Arch. f. Psychiat.*, Bd. 36, H. 2, 1902. S. 347.

THE work represented in this long paper extends over a period of five years, during which time many tabetic and epileptic patients were examined. The sense of pain was chosen as a test because it is less dependent on the will and intellectual capacity of the patient, and only one sensation was constantly investigated because of the changes that readily occur in a necessarily long *séance*, due to exhaustion both of cerebral and peripheral organs.

Alteration in the sensation of pain is in general the first and most constant change in segmental diseases of the spinal cord. Of this there may be several varieties: the sensation may be merely delayed: it may be both delayed and diminished; it may be merely diminished. A very slight delay is of importance in the early diagnosis of tabes, and the author regards it as a sign of considerable significance if the patient is unable to feel pricks of very short duration while still conscious of longer ones. The delay is well seen in an examination of the plantar reflex: the latent period is lengthened, the reflex movement lasts longer and may be followed by one or two additional movements similar to the first. Dissociation of the sensations of pain and touch is by no means uncommon. It has been found in other chronic spinal cord diseases, *e.g.* from compression: also in sulphonal poisoning and in the anaesthesia produced by Bier's method: it cannot therefore be regarded as characteristic of syringomyelia. As far as quantitative changes are concerned, the alteration occurs in the following segments in order of frequency: D 2, D 1, D 3, D 4. L 5 and S 1 are the areas in the lower extremities from which the analgesia usually spreads.

What has been said of tabes, holds true of epilepsy. After an examination of forty-four cases of true epilepsy, Jacksonian epilepsy, and hystero-epilepsy, the author finds that a varying degree of analgesia in certain areas is highly characteristic of the period before an attack. The most usually affected areas are D 2, D 1, D 3, C 8, D 4. The quality varies from the slightest hypalgesia to the most complete analgesia for pricks of long duration. General hyperalgesia usually follows an attack, seen more especially in the areas previously hypalgesic. In this connection the author lays stress on the limiting lines of the various segmental areas (the "mesial lines" of Sherrington), which are most distinctly marked where contiguous skin areas are supplied by segments which are not contiguous in the spinal cord, *e.g.* on the chest between C 4 and D 2-3. Even during the most complete

analgesia of the whole body a certain amount of sensibility remains in these lines, and the author has found that after an attack of epilepsy sensation to pain returns in these lines first. Detailed instances are given of the alteration in pain sensation before, during, and after an attack.

In tabes and in epilepsy there are certain other areas which are seldom affected in a general analgesia: a small field bounded by the middle line of the nose, the eyebrow and the edge of the jaw, corresponding to the upper part of the area supplied by the second division of the trigeminal; a field corresponding in part to the sixth cervical distribution, embracing the ball of the thumb and extending to the styloid process of the radius; and a similar small area on the sole of the foot.

The author adduces evidence to show that one is justified in foretelling an epileptic attack by the occurrence of this "prodromal analgesia." If it begins some days before an attack, the hyperalgesia that ensues will remain longer. Petit mal does not seem to have any effect on sensation, possibly because a great number of attacks follow so closely on each other.

Many questions are discussed: among them, the occurrence of spinal fits: whether the skin segmental areas are represented in the cerebral cortex: the differential diagnosis of functional and organic conditions from the point of view of the skin sensibility: the intoxication theory of epilepsy: the influence of bromide treatment on epileptic analgesia.

Photographs accompanying the paper show the author's ingenious method of representing on the skin of one individual the various alterations in the sensations of touch and pain.

Elaborate tables are given containing a concise analysis of the sensory phenomena in the series of forty-four epileptic patients.

There are copious references to the literature on the subject.

S. A. K. WILSON.

**THE POSTERO-LATERAL SCLEROSES.** CHARLES W. BURR and  
(114) D. J. M'CARTHY, *Journ. Nerv. and Ment. Dis.*, Jan. 1903, p. 14.

EIGHT cases of postero-lateral sclerosis are reported, and an attempt is made to classify combined diseases of the spinal cord.

The clinical and pathological data of these cases are well reported and illustrated. Of special interest is the account of central gliosis, which was present in two of the cases.

After commenting upon the difficulties of arranging a satisfactory classification of diseases in which lesions of the posterior and lateral columns of the spinal cord occur, the writers proceed to

emphasise this remark by classifying cases of postero-lateral sclerosis "for clinical and laboratory purposes" as follows:—

1. Friedreich's ataxia.
2. Tabes with associated diffuse sclerosis extending into the lateral columns. To this group may be added those cases of tabes associated with parietic dementia with secondary lesions in the crossed pyramidal tracts.
3. Tabes with degeneration in the crossed pyramidal and also in the direct cerebellar tracts, with or without degeneration in Clarke's column.
4. Posterior sclerosis with sclerosis of the lateral columns and disease of the anterior horns. (Chronic poliomyelitis.)
5. Primary lateral sclerosis with minor changes in the posterior columns.
6. Subacute diffuse degeneration of the spinal cord due to anæmia, cachexias, sepsis, etc., with sclerosis in the posterior and lateral tracts predominating both in clinical manifestation and under the microscope.
7. Diffuse interstitial sclerosis, seen occasionally in chronic alcoholism with multiple neuritis,\* in which the parenchymatous degeneration is secondary to the overgrowth of glial and connective tissue elements. To this same group on account of the similarity of the pathological process may be added the syphilitic postero-lateral sclerosis secondary to meningeal lesions.
8. A combined system disease of unknown origin affecting the posterior and lateral columns, and distinctly confined to the direct and crossed pyramidal tracts and the posterior columns; the direct cerebellar tract may also be involved.

The association of diseases essentially neuronc with those essentially non-neuronc, of which the causal factors in so far as they are known are distinct, and of which the clinical aspects have little in common—for example, groups 1, 2, 4 and 6, has little useful bearing. Again, upon what evidence the writers place anæmia, cachexia or sepsis in causal relation with subacute combined degeneration does not appear. The balance of evidence is strongly in favour of these conditions being unessential concomitant effects of the as yet unknown real cause.

That glial overgrowth is ever the essential cause of parenchymatous degeneration is a point as yet under dispute, and cannot be taken as proved. Group 8 contains an essential contradiction, since the regions occupied by the ventral and lateral pyramidal tract and the direct cerebellar tract have no definite anatomical limits, and moreover contain fibres other than those belonging to the systems mentioned.

JAMES COLLIER.

\* *Phil. Med. Journal*, Nov. 2, 1901.

**SPINAL HYDATID CYSTS CAUSING SEVERE "COMPRESSION (115) MYELITIS."** Operation, with Successful Results. P. TYTLER and R. T. WILLIAMSON, *Brit. Med. Journ.*, Feb. 7, 1903, p. 301.

THIS case is interesting because of the rarity of the cause of the myelitis, and also because there are very few cases of successful operation for spinal hydatid on record—the authors refer to four.

The patient was a woman, aged 27, who had a hydatid cyst removed from her back near the lower angle of the left scapula three or four years before spinal symptoms appeared. The first of these symptoms was severe pain in the mid-dorsal region of the back: a few weeks later, the legs began to get weak, and within a fortnight of this date there was complete paralysis of both legs, loss of control of bladder and rectum, and defective sensation of touch, pain and temperature on the legs and trunk up to the fifth rib; eleven days later the sensory loss was complete. A small tumour, near the third and fourth dorsal spines, was punctured and found to be a hydatid cyst.

Operation—performed about a month after the onset of weakness of the legs—disclosed fifteen hydatid cysts outside the dura mater on the posterior aspect of the cord, and they were easily scraped out.

The legs showed slight return of power and tactile sensation within a week of the operation: very gradual improvement went on, until there was complete recovery of sensation and of control of bladder and rectum, and marked recovery of motor power in the legs, which, however, soon became very rigid.

Two and a half years after the operation, the patient could perform all the coarse movements of the legs and walk alone with the aid of one stick, but there was considerable rigidity of the legs, with increase of the reflexes.

Reference is made to the two facts, important surgically, that the cysts are usually external to the dura mater, and that in most cases they lie on the posterior surface of the cord. The condition is therefore particularly amenable to surgical treatment, but *early* recognition and operation are essential in order to allow the cord to recover its functions.

ASHLEY W. MACKINTOSH.

**LEPRA UND SYRINGOMYELIE.** O. P. GERBER und RUDOLF (116) MATZENAUER, *Arb. aus dem neurolog. Institute an der Wiener Universität*, H. ix. 1902, S. 146.

ZAMBACO PASCHA in 1892 drew attention to the prevalence of leprosy in that part of Brittany where Morvan's disease was first recognised. He pointed out that some cases which had been



classed as Morvan's disease were examples of the former affection and advanced the view that syringomyelia and Morvan's disease were *formes frustes* of leprosy, a disease which at one time had probably a much wider territorial distribution, than is the case at the present day.

The anatomical examination of a large number of cases of leprosy by Laehr and others seems however to have definitely shown that the two diseases, syringomyelia and leprosy, are each associated with a distinctive morbid anatomy.

According to the authors there are only three cases in the literature, recorded respectively by Steudener, Langhans and Souza Martius where a diagnosis of leprosy was made *intra vitam* and syringomyelia found post-mortem. Gerber and Matzenauer point out after a careful review of these cases that in none was a diagnosis of leprosy justifiable on the facts.

They then proceed to describe a case, the first of its kind in the literature, in which syringomyelia and leprosy were associated. The patient, a woman of 87, presented motor, sensory and trophic changes characteristic of syringomyelia of the type of Morvan. A peculiar deformity of the nose as well as striking skin changes suggested to the authors that the case might be one of those cases of leprosy which have not infrequently been erroneously diagnosed as syringomyelia. The patient died three months after her admission to hospital. The anatomical examination showed wide spread and advanced arteriosclerosis. There was a central gliosis of the spinal cord extending through its whole length with a cavity in the cord from the level of the second cervical to the second dorsal segment.

In addition to the syringomyelia changes were found in the skin characteristic of leprosy; the bacillus of that disease being present in considerable quantities.

The authors regard the occurrence of the two diseases in the same subject as probably accidental and not etiologically associated.

EDWIN BRAMWELL.

**DIE SEELENSTÖRUNGEN AUF ARTERIOSCLEROTISCHER**  
(117) **GRUNDLAGE.** ALZHEIMER, *Allg. Ztschr. f. Psychiat.*, Bd. 59,  
1902, S. 695.

AFTER referring to Klippel's, Binswanger's and his own earlier work on arteriosclerotic brain changes, Alzheimer classifies the arteriosclerotic psychoses as follows:—

1. Arteriosclerotic cerebral atrophy.
  - a. Perivascular gliosis.
  - b. Senile cortical wasting.
  - c. Encephalitis subcorticalis chronica diffusus (Binswanger.)

2. Form with few nervous symptoms—stationary in character.

3. Arteriosclerotic epilepsy.

Etiology: same as for general arteriosclerosis.

Syphilis is very important, especially when arteriosclerosis occurs in younger subjects; alcohol less so. It also occurs as a family tendency. Senile vascular changes are not identical.

Complete arrest of the circulation is rare in ordinary brain arteriosclerosis. As a rule only a partial interference with the circulation results, sufficient to lead to a slow and partial destruction of the nerve elements, while the neuroglia hypertrophies. New capillaries and phagocytes appear as in complete softening, justifying the definition of arteriosclerotic localised lesions as *incomplete cerebral softenings*.

Diagnosis: General signs of arteriosclerosis in the heart and peripheral vessels are important, retinal changes more so. The occurrence of granular contracted kidney frequently gives valuable indications and a high percentage of the psychoses accompanied by diabetes is arteriosclerotic in origin.

The arteriosclerotic process may be confined to the cerebral vessels, the other somatic vessels being unaffected, and in some cases along with pronounced systemic arteriosclerosis the psychical disturbance is reduced to a simple paralysis or senile dementia.

Clinical diagnosis:—The mildest or nervous form (Windscheid) is characterised by rapid psychical (often physical) exhaustion, loss of memory, headache and giddiness. The condition may be fully developed by the fortieth year, the majority of Alzheimer's cases occurring between fifty and sixty-five. Hypertrophy of the heart and contracted kidney are frequently absent. The patients are often irritable and incapable of sustained application to work. Mental activity is only possible in well-worn grooves. Alcohol is borne badly.

The patient always recognises the existence of the malady, and there is frequently fear of becoming insane, but the symptoms frequently remain unaltered for years. Death generally results from apoplexy, sclerosis of coronary arteries or intercurrent affections. There is little tendency to passage into one of the more severe progressive forms.

The nerve cells in the cortex showed pronounced pigmentary atrophy, but preserved a normal structure. "Körnchenzellen" (phagocytes) were absent. Spider-cells occurred singly in the deeper layers of the cortex, and the glia was increased in the superficial layer.

The clinical phenomena, therefore, are referable to partial blood-stasis, never severe enough to lead to destruction of large areas of nerve tissue.

The second group included cases of severe progressive arteriosclerotic brain degeneration.

The disease commences with headache, giddiness and weakness of memory, with severe psychical disturbance superadded, or from the first. The patient becomes emotional, resistive and apparently apathetic. Lucid remissions quickly giving place to apathy are characteristic. The patient is very easily tired. The character is mostly a depressed, melancholic one, never exalted. Illusions and delusions of grandeur are never present according to A. Gradually the dementia becomes more and more complete, but always with the peculiarity that certain parts of the former personality remain intact for a long time.

The gradual course of the disease is interrupted by attacks of giddiness, epilepsy or apoplexy. Focal symptoms may appear afterwards, or the changes are confined to the mental area and find expression as temporary dazed conditions, unconsciousness, hallucinatory excitement and mania. The patient still recognises his own mental deficit even after speech is lost.

Such patients often present an anxious, melancholic condition, which lasts throughout the illness, although the expressions of agony become more mechanical in the later stages. The pupils seldom lose their reactions, speech is often affected. Hemipareses are not uncommon.

The age of A.'s cases lay between 52 and 64: the duration of the illness was from one to six years. Death resulted from apoplexy, heart failure, renal insufficiency, pneumonia, or diabetic coma. *Post-mortem*.—There was frequently marked diminution in brain weight, the convolutions being only slightly shrunken. The white matter is firmer than normal, with a greyish translucent colour, especially marked in the line of the vessels. Here and there miliary softenings and capillary aneurisms are met with. The *état criblé* is usually well-marked in basal ganglia and internal capsule. The grey matter of the corpus dentatum cerebelli is constantly degenerated, grey and shrunken.

Microscopically slight alterations are observable throughout the whole hemisphere. The superficial glia is hypertrophied, and the nerve cells show fatty pigmentary degeneration. Nests of spider cells, usually around vessels, occur in every section. There is, nevertheless, practically no loss of medullated fibres. Minute arteriosclerotic foci occur scattered throughout the cortex, white matter and basal ganglia, each containing in its centre a much degenerated vessel.

According to the localisation of the sclerotic process, the clinical and anatomical condition varies profoundly. The encephalitis subcorticalis chronica of Binswanger is one of the best-marked types. The long arteries of the deeper layers of the white matter

are in an advanced state of arterio-sclerosis, and the nerve fibres are correspondingly extensively degenerated, while the superficial white matter containing the short association fibres and the cortex is hardly affected. Severe arterio-sclerosis of the basal ganglia and medulla vessels is frequently associated with this (Jakobsohn).

Clinically the difficulty of connected thought is an early and striking symptom. Soon a gradual deterioration of the speech is added, frequently preceded by mere difficulty of accurate word-association. Giddiness, epileptiform and apoplectic seizures are a frequent complication, often followed by a condition of mental excitement or inco-ordination, or by the ordinary signs of focal lesions. Disturbances of speech and hemipareses may arise, gradually or suddenly, without mental change. Weeping and mechanical screaming is common. After the disease has been in existence for some time the simultaneous existence of the most varied cortical focal lesions is very characteristic. Hemipareses due to focal lesions in the internal capsule or pons are common. The pupils react promptly to the end. Speech is often markedly slower, monotonous and syllabic.

The patient is conscious for a long time of his mental defect, but ultimately falls into a dementia reminding one of animals without cerebral hemispheres (Binswanger). The duration varies from a few months to several years.

Microscopically the cortex is well preserved in striking contrast to the much degenerated white matter. There are numerous focal scleroses throughout the white matter, resulting in widespread secondary degeneration. The glia is hypertrophied throughout, and granule-laden phagocytes are numerous. Focal softenings occur in the internal capsule and basal ganglia, and very constantly in the pons in the course of the pyramidal fibres. As a result secondary degenerations are constant in the cord, usually more pronounced on one side than the other. The middle cerebellar peduncles are constantly affected, the fillet occasionally.

In senile cortical wasting the pathological process affects chiefly the short vessels from the pia to the cortex, which suffers far more than the underlying white matter. It occurs most commonly in advanced age, complicated by senile dementia (hence the name), but also may affect younger individuals. Small wedge-like foci consisting of hypertrophied neuroglia with degenerated nerve-cells and medullated fibres occurring in groups in the course of a large artery are characteristic.

*Perivascular gliosis* is another peculiar form. In this the nerve cells in the area of one or other of the main cerebral arteries are degenerated here and there as a result of a narrowing of the lumen short of actual occlusion, while the neuroglia is hypertrophied in patches along the course of the vessel. These foci

occur indifferently in cortex and white matter. When senile dementia complicates senile cerebral wasting a clinical entity arises, closely resembling that due to isolated softenings. The slow development of the deterioration, frequent slight apoplectic-form seizures, with subsequent temporary irritability, permit an accurate diagnosis during life.

Anatomically general paralysis and senile dementia are easily distinguished and diagnosed. Arteriosclerosis of the brain is characterised by the presence of foci arranged around diseased vessels, in which nerve-cells and nerve-fibres are degenerated, the neuroglia hypertrophied, and "Körnchenzellen" are practically constant. Wide-spread secondary degenerations are common, but beyond these and the primarily affected areas the brain is practically normal, and such normal areas occur in even the severest cases. This is in marked contrast to the essentially diffuse processes characteristic of dementia and paralysis.

Clinically this difference in the fundamental lesions expresses itself in a characteristic form of dementia. One of the earliest symptoms to appear, and one which often remains even in the latest stages, is the remarkable slowing and laboured character of the mental processes, indicating interference with the association faculty. An actual depressed condition is not associated with it, rather a feeling of helplessness and indecision. The drowsy, dreamy state of paralytics and demented is here absent. The rapidity with which such interference arises, disappears and again appears is characteristic. The narrowing of the patient's interests occurs here as in paralysis and dementia, but the consciousness of personality remains much longer. The affections become gradually dulled, but continue normal in character.

The psychical deficiencies are more profound, but also more circumscribed, than in dementia paralytica or senilis. Apart from attacks of irritability the essential phenomena in arteriosclerotic atrophy are the direct effects of the focal lesions, whereas in senile and paralytic dementia the abnormal mental phenomena, excitement and delusions are more prominent.

The greatest difficulty in differential diagnosis is presented by Lissauer's atypical paralysis and certain forms of senile dementia with focal lesions. Senile dementia with focal lesion has the same relation to pure senile dementia that Lissauer's paralysis has to general paralysis. The process consists in a specially severe localisation of the senile degeneration in circumscribed areas, especially of the posterior lobes, extensive degeneration of medullated fibres and great dilatation of the ventricles. Clinically epileptiform and apoplectic seizures, well-marked focal lesions varying with the site of severest degeneration are superadded to the ordinary signs of senile dementia. The condition has no

connection with arteriosclerosis. Lissauer's paralysis is a true paralysis affecting the whole brain, although more advanced in a restricted area. The dementia is of the paralytic type. The condition of pupils, speech and reflexes also assist the diagnosis.

*Arteriosclerotic epilepsy*: Two forms. First or cardio-vascular form. Epileptiform seizures occur in the subjects of severe atheroma of the vessels and disease of the heart, and seem to be closely related to the disturbance of the circulation. Psychical defects may be absent. Naunyn showed that by compression of the carotids in arteriosclerotic subjects, epileptiform seizures could be induced. In one of A.'s cases the face was intensely pale during the attacks. The attacks often disappear under the administration of digitalis, and reappear when it is discontinued.

In the second form the attacks are associated with focal symptoms, *e.g.*, a patient after an attack presented weakness of the right hand and slight sensori-motor speech defects. Post-mortem arteriosclerotic foci were found on the left post. Rolandic and parietal lobes. The brain seemed otherwise healthy (Jacksonian type).

A. cannot regard the epilepsy of old acoholics as due to arteriosclerosis, and considers that it is due to brain changes induced by the alcoholic poisoning.

In the discussion which followed, Professors Fürstner, Degenkolb and Hoeneel took part.

J. A. MURRAY.

**ÜBER DIE ERKRANKUNG DER HINTEREN WURZELN MIT  
(118) FEHLEN DER KNIESEHNENREFLEXE BEI HIRN-  
TUMOREN. ERBSLÖH, *Monatssch. f. Psychiat. und Neurol.*,  
Feb. 1903.**

THIS paper deals with the lesions of the posterior roots, and of the posterior columns of the cord which are found in a large percentage of cases of cerebral tumour, and which are somewhat analogous to the lesions which are seen in the same regions in *tabes dorsalis*.

The author gives a description of two cases of cerebral tumour, in both of which the classical signs were well marked, and in both the knee-jerks were much diminished.

Post-mortem a tumour was found in the left motor region in one case, and in the temporal lobe in the other. In sections of both cords, stained by the Marchi method, numerous degenerated fibres were seen in the intra-medullary position of the posterior roots. This degeneration consisted of an atrophy of the myeline sheath, and in one case the axis cylinders were also affected.

In the posterior roots in both cases at the level where the roots

acquire a sheath from the pia mater and the sheath of Schwann appears, the fibres showed a more advanced degree of degeneration than was seen in the intra-medullary portion, and the author concludes that the lesion started here, and that the changes seen in the cord were secondary to it. These changes did not spread to the ganglion; nor did the cells of the posterior root ganglion show any sign of injury.

The fibres of the anterior roots were also affected, but to a less degree.

In discussing the cause of the nerve root being attacked in these cases he rejects the toxic and the mechanical theories which have been advanced by other authors, as well as the theory that it is due to a strangulation of the root at the level of the ring of Redlich and Obersteiner.

As a possible explanation, he suggests that as the extra-medullary portion of the posterior root has a poorer blood supply than the other parts of the cord, it would react first to a disturbed circulation such as may be caused by a cerebral tumour, and he therefore attributes this degeneration to a disturbance of nutrition.

R. G. Rows.

**DIE BEZIEHUNGEN DES STIRNHIRNS ZUR PSYCHE.**  
(119) E. MUELLER. *Allg. Ztschr. f. Psychiat.*, Bd. 59, 1902, S. 830.

IN agreement with Bruns, Mueller criticises adversely the evidence adduced, especially by E. Welt, of the localisation of the mental functions in the frontal lobes, as the result of a careful analysis of 164 cases from the literature (including a case of his own). In pointing out the fallacies in the observations he comes to the conclusion that a definite judgment for or against cannot so far be arrived at.

Tumours of the frontal lobes, from their frequent large size, have a great tendency to excite general symptoms and mental changes can only be regarded as of importance for localisation when post-mortem evidences of general action are absent or where during life they preceded by a considerable interval the symptoms obviously referable to the general action of the tumour. The same criticism applies to experiments on animals.

The dementia described in cases of frontal tumours is very often really a mental confusion of slight degree. There is a tendency to underestimate the importance of the neuro—or psychopathic diathesis in the etiology of frontal tumour. Hereditary taint (including foetal diseases and injuries during parturition), trauma, alcohol and syphilis, which all act as predisposing causes to tumour, are also the main factors in producing the neuro- or psychopathic diathesis, and in *e.g.* a case of tumour with psychosis

following trauma, the trauma is not only of importance in the etiology of the tumour but also may have given rise to the tendency to the psychical disturbance elicited by the general action of the tumour.

It is therefore necessary to exclude for local diagnostic purposes cases in which the neuro-psychopathic predisposition exists. There are three relations to be considered as possible in the existence of tumour and psychosis. (a) The tumour may be the prime factor inducing mental symptoms in a previously healthy brain (general action) or eliciting or intensifying a psychosis in a predisposed patient. (b) Tumour and psychosis may develop on a common basis of congenital or acquired predisposition. (c) A pre-existing psychosis may favour the development of a tumour.

Various authors, especially Gianelli, have maintained that only frontal tumours produce from the first psychical changes. Mental affections, however, are not uncommon as initial symptoms in tumours in other regions, especially in the predisposed and they can only be regarded as focal symptoms if they were frequently observed as early symptoms in non-psychopathics and always preceded the general symptoms of tumour. In the absence of focal symptoms the mental disturbance is more striking and in tumours of other regions the focal symptoms divert the attention of the clinician from slight mental aberrations which may exist. Deducting those cases in which the mental symptoms do not precede the general classical signs of tumour only 25 per cent. of cases would come under consideration and of these nearly one-half occur in psychopathics.

An analysis of 25 cases of bilateral tumour shows that psychical disturbance is not a constant and rarely an intense symptom—and in many cases only the confusion common to all brain tumours is observed.

The rapid improvement in the mental condition following operation is strongly in favour of the view that the mental disturbance is a general symptom comparable to vomiting, choked disc, etc., rather than a focal one. Cases in which the tumour has not been removed, the dura not being opened, may present marked mental improvement; this, the author believes, is quite incompatible with the mental defect being a focal symptom. The view that the changes in animal experiments after removal of portions of frontal lobes are due to alterations in the remaining portions of nerve tissue is in the case of complete removal of the frontal lobes an argument for the thesis which M. upholds.

The differential diagnosis of the slight degrees of mental confusion seen in tumour from dementia is sometimes difficult and a frequent source of error. The main characteristics are, difficulty of perception, long reaction time, rapid exhaustion of the mental



processes on continued examination, and especially the marked variations in degree of intensity of the various symptoms. The correct answer may be elicited after several repetitions of a question. At times the patients are conscious of their condition and at others not. Real loss of memory is only for recent events. Grave psycho-motor disturbances are frequent, stupor and apathy or restlessness and incoherence. Control of sphincters is lost only in severe cases.

Friedmann's experiments on diffuse cortical changes as a result of punctured wounds of the cerebrum are referred to as showing the importance of a general microscopical examination of brain areas remote from the tumour, and suggesting an explanation of the diffuse cortical changes in the severe chronic nervous sequelæ of concussion. The paper concludes with an appeal for more accurate clinical and pathological examination of psychoses occurring in cases of cerebral tumour.

J. A. MURRAY.

**ON AMAUROTIC FAMILY IDIOCY, A DISEASE CHIEFLY OF  
(120) THE GREY MATTER OF THE NERVOUS SYSTEM.**

B. SACHS. *Journ. Nerv. and Ment. Dis.*, Jan. 1903, p. 1.

THE author, by whom the clinical picture of amaurotic idiocy was first described, records the clinical history and the pathological anatomy of another case. The description of the changes found in the nervous system is admirably described and is well illustrated.

The case was that of a child aged two years and eight months. The parents were Hebrews. Of three children, the eldest was healthy at the age of six years; the second died blind and paralysed at the age of sixteen months; the third is the case recorded. This child was never able to sit up. Vision was noticed to fail at ten months, and complete blindness was present at the age of one year, when the typical changes at the macular region were observed. At nineteen months convulsions commenced, and general contractures ensued. Death occurred from broncho-pneumonia. The brain presented the characteristic walnut-like appearance, and weighed 784 grammes. There was deficiency in the development of the cerebral white fibres. Systemic degeneration was present throughout the pyramidal system, and also in the ventro-lateral region of the cord.

Most remarkable cell changes were found throughout the whole grey matter of the brain and spinal cord; and in the cerebral cortex and ventral horns of the spinal cord not one normal nerve cell was found. The nature of the changes was the same in all the cells, which were much swollen and their contour completely altered. The protoplasm was in a condition of more or less com-

plete disintegration, a homogeneous mass resulting. The nucleus was often absent, sometimes in the process of being extended, and when present always excentric and without definite outline. A little tigroid substance was present in the cells of the spinal ganglia. Vascular and glial changes were not present. These results are in entire agreement with those of Hirsch.

The author, who in his earlier communications was inclined to regard the condition as one of cerebral agenesis simply, is now convinced that it is a degenerative condition as has been also held by Kingdon and Russell, Hirsch and others. He thinks, however, that such degeneration does not necessarily represent an acute or an acquired affection. He contends that wherever normal growth is arrested degeneration sets in. The child afflicted with amaurotic idiocy is endowed, as regards its nerve elements, with a certain brief tenure of life only, upon the lapse of which degeneration occurs. The author suggests the term "abiotrophic" (Gowers) or "abiotic" as a convenient term for the description of such a disease. He rejects a toxic cause for the disease, and dismisses Hirsch's contention that a toxic influence could have been exerted after birth through the mother's milk, since several of his patients were not suckled by the mother.

Attention is drawn to the remarkable disparity between the universal cell degeneration and the much less marked degeneration of medullated fibres in the nervous system.

Certain details of morbid anatomy in cases described by Spiller, Rolly, Collier, Myer, and Levi are given. The author refutes the imputation of Peritz that he has made a special effort to establish this condition as an entirely independent disease, and states that he has only contended that this family disease represents a distinct and easily recognisable clinical type. He further urges that the clinical type as now accepted be not disturbed by the inclusion of unusual, atypical and complicated cases.

A considerable bibliography is appended, including a reference to an analysis of 64 cases by Falkenheim (*Jahrbuch f. Kinderheilkunde*, N.F., liv., H. 2, 1901).

JAMES COLLIER.

**UN CAS D'HYSTÉRO-SYPHILIS.** S. THOORIS, *Journ. de Neurol.*, (121) Jan. 1903, p. 69.

In this paper the author describes the following case:—

A man, whose previous health had been excellent, contracted syphilis at the age of 22, and while suffering from the secondary manifestations of the disease he developed a left hemiplegia. In spite of anti-syphilitic and electrical treatment his condition remained unchanged for four years. When examined he was

found to have a left hemiplegia and a complete left hemianesthesia. Except for the pupil reflex, which was normal, all the superficial and deep reflexes were abolished on the left side. The left visual field shewed concentric contraction. His mental condition was one of slight exaltation with impairment of memory. Three weeks later, though under no anti-syphilitic treatment, recovery was complete, save for the amnesia, which had somewhat increased. An analysis of the cerebro-spinal fluid gave a negative result. The author concludes that the patient was suffering from a non-organic nervous disease, which he calls "Hystero-Syphilis."

T. GRAINGER STEWART.

**A CASE OF GASTRIC DILATATION AND TETANY. RECOVERY.**

(122) W. E. CARNEGIE DICKSON, *Practitioner*, Jan. 1903, p. 44.

THE patient, a tailor, aged 47, was admitted into Professor Greenfield's wards in the Edinburgh Royal Infirmary on 3rd October 1901. He had suffered from stomach trouble for sixteen or seventeen years, and came under treatment for gastric dilatation and atonic dyspepsia. He had never previously suffered from tetanoid complications. On examination he was found to present the typical clinical picture of old-standing torpid or atonic dyspepsia with gastric dilatation and retention, probably due to some form of pyloric obstruction. The lower border of the stomach was two inches below the umbilicus; succussion showed the presence of a large amount of fluid in the organ; and marked peristaltic waves occurred from time to time, emerging from under the left costal margin and travelling downwards and towards the right. On these occasions the thickened pylorus could be distinctly felt in a state of spasmodic contraction. For a month after admission the patient improved under ordinary treatment, but on October 29th he vomited a large quantity of clear, almost colourless fluid. On November 1st at 9.30 P.M. he again vomited and complained of a tingling sensation in the skin of the hands. A repetition of these symptoms occurred on the following morning at 1 and again at 3.30 A.M., the latter attack being immediately followed by the tetanoid seizure. The latter in most of its details corresponded with the classical descriptions of the disease. The hands and forearms were the parts chiefly affected, the symptoms being much more marked on the left side. There was alternate involvement of the extensor and the flexor muscles of the forearm, whilst the fingers at one time presented the typical "accoucheur's hand," at another the "main en griffe" position. The muscles of the upper arm were also implicated, but those of the lower extremities were scarcely, if at all, affected. The facial muscles, and those of the

eye, tongue, cheek, neck and trunk were also involved with varying intensity in the subsequent attacks, as were also the functions of speech and sight. Trousseau's phenomenon (*i.e.* recrudescence of the muscular spasm on pressing over the main nerves or blood-vessels of the part) was well marked. The pupils at the onset of tetany were widely dilated and immobile, but later were semi-contracted and reacted to light only with great sluggishness. Consciousness was not lost, but the patient usually became very collapsed, and at times seemed almost moribund. The muscular contractions were exceedingly painful, but the patient obtained some relief on the contracted muscles being firmly kneaded, and the tetanoid spasms actively resisted and counteracted. The heart's action became very rapid and feeble, and the pulse was often impalpable, though the vessel wall could be felt apparently in a state of spasmodic contraction during the acute attacks. The stomach contents varied considerably in character from time to time. They were exceedingly foul-smelling, and contained large numbers of sarcinae at first, and later, considerable numbers of bacilli, probably varieties of *B. Coli*. Attempts to isolate any toxin from the stomach contents were not successful, nor did culture and inoculation experiments lead to any definite result. The stomach itself, during the attack, underwent very extreme dilatation or distention, on one occasion reaching almost to the symphysis pubis. No urine was passed for a period embracing the twenty-four hours before and sixteen hours after the onset, at the end of which time sixteen ounces containing a trace of albumin were withdrawn by catheter. During, and for a considerable period after the attacks, there occurred a leucocytosis of 17,500 to 40,000, mostly polymorpho-nuclear in character—a very strong argument in favour of the theory that the disease is toxic in origin. The treatment adopted consisted in the frequent and thorough washing out of the stomach; the exhibition of sodium phosphate and later, of carbolic acid for gastric condition; bromide, and small doses of erythrol tetranitrate for the muscular and vasomotor spasm; stimulants, such as strophanthin, ether and brandy for the collapse, together with saline transfusion and enemata. After the active attacks of tetany had passed off, the patient gradually sank into an apparently moribund condition, and on November 13th was almost insensible. Even nutrient enemata were not retained, and he required constant hypodermic stimulation. He remained in this condition for the next two days, becoming delirious and requiring constant watching. On November 16th and 17th, however, some improvement occurred, and thereafter he made a slow but very satisfactory recovery, leaving hospital on January 18th, 1902. He returned to the ward on May 30th for observation, and was found to be in a fairly good state of health. On June 10th, however, he



had an attack of cramp in the lower extremities, and, in view of a possible recurrence of the tetany, the operation of posterior isoperistaltic gastro-jejunostomy was performed by Mr Francis Caird. The pylorus was found to be greatly narrowed, somewhat thickened and hypertrophied, and there were firm, puckered, cicatricial adhesions between it and the gastro-hepatic and great omenta. After the operation the patient made a rapid and uninterrupted recovery, and is now in very good health, a result which, when taken in conjunction with the three successful cases recorded by Mayo Robson, certainly justifies active surgical interference in cases of gastric tetany.

AUTHOR'S ABSTRACT.

**EINE MERKWÜRDIGE COMPLICATION EINES MIGRÄNE-**  
(123) **ANFALLES.** L. HOEFLMAYR, *Neurol. Centralbl.*, 1 Feb. 1903,  
S. 102.

THIS is a record of a remarkable case of migraine. The patient was a woman, 57 years of age, whose father and sister both suffered from this complaint. She herself had had attacks of varying severity for the preceding 40 years. In the attack which forms the subject of the paper the symptoms began in the usual way with headache, and there was also spasm of one eyebrow and eyeball. Then sickness came on and recurred several times. This passed off on the following day, but the headache persisted in spite of treatment. That evening she became apparently unconscious. The writer of the paper saw her on the following day. For four days after this she remained in an unconscious state, but took fluid food reflexly. Then improvement began to set in, and she gradually came round. There was then discovered to be a scotoma of the field of vision, and this persisted more or less for a month. She had no recollection of events which happened during her period of unconsciousness. The possibility of hysteria or simulation is rejected. The most feasible explanation of the case, in the author's opinion, is that it was due to a toxine. Before the attack she had suffered from very severe constipation, and had also had serious mental strain.

JAS. MIDDLEMASS.

**RHEUMATOID ARTHRITIS AS A CEREBRO-SPINAL TOXÆMIA.**  
(124) R. LLEWELYN JONES, *Edin. Med. Journ.*, Jan. 1903, p. 25.

UNTIL recently there have been two distinct theories as to the etiology of rheumatoid arthritis—the nervous and the infective. The author is of opinion that both are correct, the organism being the primary cause and the nervous system the medium through which the clinical phenomena are coloured. The vasomotor disturbances preceding and accompanying the joint affections are

indistinguishable from Raynaud's disease. Patients may suffer for years from Raynaud's disease, asthma, or migraine before the appearance of fusiform articular swellings, or, on the other hand, the sequence may occur in a few weeks.

On consideration of cases in which the ring and middle fingers are affected, it is observed that the local syncope, cramps and irritability of the flexor muscles, periarticular swellings, œdema, and sweating and sensory disturbances of particular areas of skin could all be accounted for by an affection of the 8th cervical territory (Head). The cramps and myotatic irritability seen at this early stage have a great similarity to those occurring in tetany; moreover Chvostek's symptom may be present, and the electrical reactions often show a sluggish faradic and brisk galvanic response with A.C.C. equal to K.C.C. The periarticular swellings subside in the intervals between the paroxysms and the different phenomena rise and wane concomitantly. Very soon, however, this passing functional disorder gives place to the stage of structural alteration. Trophic changes occur in the skin and these may correspond to a spinal area. Muscular atrophy and contractures appear, and in some cases paralysis, with loss of deep reflexes. Articular affections have been observed in Raynaud's disease, and the joint changes in rheumatoid arthritis are probably vasomotor in origin. The author has seen symmetrical gangrene in rheumatoid arthritis. Uniform contraction of the field of vision is common and is probably due to vasomotor disturbance of the retinal vessels. Optic atrophy, hippus, ptosis, sluggish action of the pupil, and retraction of eyeball have all been noted. A close connection seems to exist between rheumatoid arthritis and Graves's disease, and it is possible that the increased pulse rate in some cases of rheumatoid arthritis may be due to exophthalmic goitre. Some forms of tetany are like the condition which follows extirpation of the thyroid. In some cases of rheumatoid arthritis preceded by Graves's disease atrophy of the thyroid has been observed. Thyroidectomised patients benefit from thyroid extract, and considering the intractable nature of rheumatoid arthritis the writer thinks it would be justifiable to try the effect of thyroid extract in such cases as the above.

HENRY J. DUNBAR.

**CONGENITAL DEFECT OF ABDUCTION ASSOCIATED WITH  
(125) RETRACTION OF THE EYEBALL IN ADDUCTION.**

JAMESON EVANS, *Ophth. Rev.*, Jan. 1903, p. 1.

In this interesting paper the author gives notes of two cases which have come under his personal care, and appends a summary in tabular form of twenty-seven cases, collected from the literature

of the subject. The most prominent features of the clinical picture are indicated in the title, and are further summarised as follows.

The condition is congenital. In the primary position the axis of the affected eye is either parallel with that of the other eye, or may show slight convergence or divergence.

In the majority of cases the defect is confined to one eye, and there may be slight enophthalmos, with narrowing of the palpebral fissure.

When the patient endeavours to carry out lateral movements of the eyes, the more distinctive features become apparent. On attempted conjugate movement towards the unaffected side, there is imperfect inward rotation of the affected eye, with retraction into the orbit, and consequent narrowing of the palpebral fissure. There may also be upward or downward movement of the globe.

When, on the other hand, conjugate movement is attempted towards the affected side, the defective eye returns to the primary position with forward movement of the eyeball, and expansion of the palpebral fissure. The outward movement is not carried beyond the vertical mesial plane of the orbit. When the effort of convergence is made, the normal eye is turned inwards and the affected eye carries out the same movement as in attempted conjugate deviation to the affected side.

The pupils do not contract when convergence is attempted. The vision of the affected eye is seldom full, and sometimes highly amblyopic. Diplopia may be entirely absent, or may only appear on fixation in certain directions. Headache is seldom complained of. The condition rarely occurs in more than one member of the same family. It is more frequent in females, and much more frequent on the left side.

In discussing the pathology, the author refers to two theories of explanation advanced by Türk. According to the first—the theory of faulty insertion—the internal rectus which causes retraction of the globe is inserted further back than normal. The portion of the muscle capable of unwinding itself from the globe is thus diminished, and internal rotation is correspondingly replaced by backward traction of the globe.

The second—the fixation theory—explains the condition by reference to the fibrous and inelastic state of the external rectus, in which the muscle fibres have been largely replaced by connective tissue. The action of the internal rectus thus fails to produce internal rotation, but results in retraction of the eyeball.

Evans is in favour of the first theory, but also thinks that excessive fibrous change, or shortening of the muscles often plays a part.

In proceeding to treatment, Evans suggests the use of traction forceps to test the internal and external rotation.

The tendons of the internal and external recti are then to be exposed, and their insertions examined. If the insertions of these muscles are found to be normal, and no accessory bands are discovered further back, the symptoms may be considered to be due to a fibrous external rectus, and dealt with accordingly. If, on the other hand, the insertions are found to be too far back, with accessory bands present, lengthening as well as advancement of the internus is recommended.

A. H. H. SINCLAIR.

**UNILATERAL OPHTHALMOPLÉGIA EXTERIOR AND INTERIOR.**

(126) FERRON, *Ann. d'Ocul.*, 1902, p. 351.

THE theory currently accepted appears to be that ophthalmoplegia purely interior or purely exterior is indicative of a nuclear situation of the causative lesion. This opinion Ferron assails with much earnestness from the anatomical as well as from the clinical point of view. He makes much of the difficulty so hard to explain away of the crossing of the fibres of the fourth nerve: one instructive indication of the extreme seriousness of this difficulty with which however Ferron does not deal being the fancifulness of the explanations advanced to dispose of it. The puzzle is how to explain a lesion of the third and sixth nuclei on one side along with paralysis of the fourth nucleus on the opposite, and an intact state of the fourth on the same side as the sixth and third. This is required to produce, according to the usual opinion, a purely unilateral ophthalmoplegia. Besides this it is necessary that only certain of the cell-groups in the third nucleus should be affected, while others are left normal. Ferron complains that in order to be able to make neat diagrams with nuclei partitioned off into little areas observers have somewhat wrested facts. It is convenient to show the nuclei of one third nerve, for example, all on one side of the middle line, though in point of fact the balance of evidence points distinctly the other way. Grasset thinks that in the motor path connected with the eye there is a chiasma, just as there is in the visual, and that from each hemisphere there arises a hemi-oculo-motor nerve analogous to the half-vision centre.

The difficulty in the way of acceptance of a basal situation of the lesion in such cases has been that it is not easy to conceive how in the trunk of a nerve some fibres should be completely thrown out of function by an external lesion while others are left entirely uninfluenced. But after all is it any more difficult to conceive how different fibre-groups are attacked or spared than different cell-groups in the nuclear area? As a matter of fact two



cases have been carefully observed *intra vitam* in which, while some of the muscles supplied by the third nerve have been paralysed, others have altogether escaped, and yet in which post-mortem examination corroborated clinical evidence that the situation of the lesion was basal. Several examples of this state of affairs are given in detail by Ferron, whose conclusion is to the effect that ophthalmoplegia exterior (or interior) is not a nuclear lesion and may be a basal one. An interesting confirmation of this view appears too in Von Graefe's *Archiv für Ophthalmologie*, lv. 2. In a previous number of that journal Salomonsohn had given a long and careful article on a case of ophthalmoplegia, showing how the lesion must probably be entirely peripheral, but in the later issue he has to withdraw from this conclusion since the further advance of the case seemed to prove beyond a doubt that there was a neoplasm at the base which was responsible for the paralysis. Both the French and the German articles are worthy of careful study.

W. G. SYM.

**UEBER EINE EIGENTHÜMLICHE SCHREIBSTÖRUNG. MICRO-  
(127) GRAPHIE, IN FOLGE CEREBRALER ERKRANKUNG.**

A. PICK, *Prag. Med. Wochensch.*, xxviii., Nr. 1, 1903.

THE author here relates the symptoms in two cases which exhibited a peculiar alteration in writing. The first was a workman, 36 years of age, who had an apoplectic stroke with subsequent impairment of memory. After death there was found a chronic meningitis, brain atrophy, syphilitic arterial disease, and a number of small softenings in both hemispheres. When his writing was tested it was found to be correct in form but exceedingly small in size, though there was no sense in the words written. The second was that of a foreman, 27 years old, who, during his military service, contracted syphilis. He was seized with hemiplegia and loss of speech, but these gradually passed away. During recovery his writing was observed to be exceedingly small, even when copying. He was unable to explain why he wrote in this way. As an explanation of this curious change the author inclines to the view of Embden, who had observed a similar condition in chronic manganese poisoning. It is considered to be due to an increase of muscular tension. Samples of the writing in the two cases are given.

JAS. MIDDLEMASS.



**NOTE SUR L'ÉTAT DES RÉFLEXES CUTANÉS ET PUPILLAIRES  
( 128) ET DES SENSIBILITÉS TESTICULAIRE ET ÉPIGAS-  
TRIQUE PROFONDES CHEZ LES DIABÉTIQUES. M. A.  
PITRES. *Extrait des Comptes rendus des séances de la Réunion  
Biologique de Bordeaux. (Séance du 11 novembre 1902).***

BOUCHARD and others have shown that the knee-jerks are lost or very feeble in 40 per cent. of individuals suffering from diabetes mellitus.

The author has examined certain reflexes in thirty-two cases of this disease and tabulates his results as follows:—

REFLEXES.	KNEE.	ABDOMINAL.	CREMASTERIC.	PLANTAR.	PUPIL.
Abolished -	13 times	16 times	19 times	16 times	1 time
Feeble -	7 „	8 „	6 „	2 „	0 „
Exaggerated	2 „	6 „	4 „	6 „	0 „
Normal -	10 „	1 time	3 „	8 „	31 times

In not one of the thirty-two cases was there true testicular or deep epigastric analgesia. Testicular and deep epigastric analgesia as well as the Argyll-Robertson pupil may be valuable signs in distinguishing true tabes from diabetic pseudo-tabes.

EDWIN BRAMWELL.

**SUL RIFLESSO SCAPOLO-OMERALE. G. ESPOSITO, *Manicomio*  
(129) Anno xviii., No. 3, p. 437.**

ESPOSITO records a large number of observations made by him on the subject of Bechterew's scapulo-humeral reflex. He employed as his material 32 healthy persons, 176 patients suffering from various forms of mental disease in the asylum at Nocero, and 5 cases of nervous disease, including pellagra. In health he never failed to elicit the reflex, and of the 176 mental cases it was present in all save 2. In the 5 cases of nervous disease the scapulo-humeral reflex corresponded with the other tendon reflexes as to exaggeration or abolition. Esposito therefore concludes that the scapulo-humeral reflex is one of remarkable constancy, and that it is of the greatest importance in coming to an opinion with regard to the condition of the reflex arc in the upper extremity.

He vigorously criticises the results of Haenel, who examined 120 persons free from nervous disease, and was able to elicit a reflex in only 43 per cent. of them. Esposito points out that Haenel's observations were made by percussion merely at the lower angle of the scapula, instead of at the spot accurately described by Bechterew, viz.:—the inner edge of the bone near its lower angle. He then goes on to discuss the nature of the scapulo-humeral phenomenon, and concludes that it is a true reflex, elicited from stimulation of the periosteum or fascia of the scapula. The complicated nature of the reflex movement shows that it cannot be accounted for by any mere mechanical stimulation of muscle fibres, inasmuch as the resultant movements consist in adduction and slight external rotation of the humerus (infra-spinatus and teres minor) and sometimes, though less frequently, abduction of the arm and slight flexion of the elbow (deltoid, biceps and brachialis anticus)—a combination too complex to be accounted for by any other hypothesis than that of a true reflex. This reflex is exaggerated in those cases with increase of other tendon reflexes, and diminished or lost in lesions of the lower motor neurone corresponding to the 5th and 6th cervical segments of the cord.

PURVES STEWART.

**NOTE À PROPOS DU RÉFLEXE DE BABINSKI.** C. HELDEN-  
(130) BERGH, *Journ. de Neurol.*, Jan. 1903, p. 71.

In a previous paper the author pointed out that in addition to the normal skin reflexes, there was a series of abnormal skin reflexes, of which Babinski's reflex is an example. He seeks to classify the reflexes according to the tracts which are concerned in their production. This classification is based upon the fact that if the pyramidal tract is seriously affected the result is exaggeration of the tendon reflexes, diminution or abolition of the normal skin reflexes, and the presence of an abnormal skin reflex, as seen by the occurrence of Babinski's sign, from which the author concludes that the tendon reflexes and the abnormal skin reflexes pass by the extra pyramidal tracts, while the normal skin reflexes pass by the pyramidal tracts. He furthermore points out that the extension of the great toe is not a cortico-spinal reflex, but a spino-mesencephalic reflex. In support of this view he cites two cases; the first recorded by Homburger, the second mentioned by M. Dejerine. Of these, the latter does not appear to be applicable to his argument.

T. GRAINGER STEWART.

**PSYCHIATRY.****DIE DIAGNOSE DER PROGRESSIVEN PARALYSE. NISSL.**

(131) *Versamml. d. S. W. deutsch. Irrenärzte.*, Nov. 1902. *Neurol. Centralbl.*, 16 Dec. 1902, S. 1151.

NISSL states it as his opinion that the diagnosis of general paralysis is made much too often. He regards it as belonging to the group of inflammatory processes, when all the pathological changes associated with the disease are taken into account. Further, since the clinical diagnosis is by no means always certain, it would mark a step in advance if one were able to separate on clear pathological grounds cases of paralysis from all other cases of brain disease. He goes on to discuss the ectodermal and mesodermal constituents of the brain. The vessels are mesodermal in origin, and everything outside them is ectodermal. Many experiments prove that the ectodermal constituents of the brain exercise a restraining influence on the growth of the mesodermal, and conversely. The same is true with regard to the nervous and non-nervous ectodermal elements. If the restraining influence of the former over the growth of the latter is removed, these grow and not the mesodermal constituents. But the result is different when the whole of the ectodermal tissues is destroyed, as, for example, by a hæmorrhage. In that case the mesodermal constituents begin to grow, vessels sprout out, endothelial cells develop into fibroblasts, and with these the granular cells are also formed. This distinction is perhaps most clearly seen in granulation tumours, which are composed entirely of mesodermal tissue. It is absolutely necessary to bear this distinction in mind if the real nature of the histo-pathological processes in the central nervous tissue are to be rightly understood.

Nissl has already drawn attention to the peculiarities of purulent meningitis, and thinks he has shown that leucocytes never diapedese from the vessels as they do in other organs and form a purulent infiltration. In the brain the only characteristic exudate is the cellular infiltration of the adventitial sheath by the plasma cells of Marshalko. In the presence of this exudate we possess a certain criterion of inflammatory processes in the nervous system.

Up to now two forms of inflammation of the brain are known—(1) those spreading from the meninges, and (2) those which are autochthonous. Under the latter come (a) acute and non-purulent encephalitis and myelitis, and (b) chronic inflammation of the central nervous tissue, especially of the cortex. To the last belong all doubtful cases of paralysis. From this it follows that one group of psychoses can undoubtedly be separated from all others, on the basis of histo-pathological observation, and that to this one group all doubtful cases of paralysis without exception

belong. This group shows infiltration of the adventitial sheaths with Marshalko's plasma cells.

This infiltration is not, however, characteristic of paralysis. It only characterises the inflammatory nature of the process which is the basis of the paralysis. It thus affords even inexperienced persons a means of determining the presence of chronic inflammation of the central nervous cortex. JAS. MIDDLEMASS.

**L'ASSISTENZA DEGLI ALIENATI E IL PATRONATO**  
(132) **FAMILIARE IN ITALIA.** A. TAMBURINI, *Riv. speriment. di freniatria*, f. 4, 1902, p. 671.

**DES MOYENS D'AMÉLIORER L'ORGANISATION MÉDICALE DES**  
(133) **ASILES EN BELGIQUE.** J. CROCQ, *Journ. de Neurol.*, 5 jan. 1903, p. 1.

**CONGRESSO INTERNAZIONALE DELL' ASSISTENZA DEGLI**  
(134) **ALIENATI E SPECIALMENTE DELLA ASSISTENZA**  
**FAMIGLIARE.** (Anversa, 1-7 Settembre 1902.) G. C. FERRARI, *Riv. speriment. di freniatria*, f. 4, 1902, p. 737.

THE meeting of the *Congrès International de l'Assistance des Aliénés et spécialement de leur Assistance Familiale*, held at Antwerp in September last, was undoubtedly an event of very considerable importance to the department of practical medicine to which its deliberations related. It was attended by alienists fully representative of the best Continental opinion in their specialty, several very able papers were communicated to it, some of which gave rise to extremely animated discussions, and the resolutions adopted, the authoritative value of which cannot be impugned, sharply conflict in several particulars with the common practice, and certainly not least with that of our own country. There is, moreover, a very special reason why we should be interested in what took place at Antwerp on this occasion, in as much as it is announced that the next meeting of the Congress is to be held in Edinburgh in 1904.

It is intended to notice here the papers that were read by Professor Tamburini and Dr Crocq, and the very full and interesting account of the meeting that has been given by Dr G. C. Ferrari.

Italy, like many other countries, is feeling the pressure of an increasing burden of lunacy. Tamburini states that recent enquiries have shown that there is in his country an important progressive increase in the number of the insane, a serious and growing state of overcrowding of the asylums, and a relative as

well as actual increase in the cost of maintenance to the provincial administrations. The many remedies for overcrowding that have been suggested and tried have so far proved insufficient. In 1891, in conjunction with Lombroso, he submitted to the Italian Government a special report on the asylums of the State, and made certain proposals which, although received with much favour, have never become law. The introduction of improvements has consequently been left to the initiative of the provincial administrations and directors of asylums. Much has been done to relieve the pressure upon the asylums by placing certain classes of patients, such as quiet demented, imbeciles and the pellagrous, in other institutions. Something has also been effected by the development of the system of family care of the insane. In 1898, about 4 per cent. of the insane were under this system, and the proportion has slightly increased since. The majority of these patients are boarded with their own relatives (*custodia domestica*), the rest in the families of strangers (*patronato familiare*). The latter is an adaptation of the independent colony system of Gheel and Lierneux. The patients are visited regularly by one of the doctors of the asylum with which they are connected. Tamburini, on the ground of his own experience of it, very strongly recommends the adoption of this system for certain classes of patients. He is of opinion that the independent colony system and the Scottish private dwelling system have each their own advantages and inevitable inconveniences, and that both deserve to have a place in a complete system of provision for the insane. He believes, however, that the system of family care of the insane best adapted for Italy is the mixed one that he has employed, which consists in the boarding of the patients with families in villages near to the asylum, under the supervision of the asylum superintendent. Whilst he advocates the adoption of this plan, he looks upon it as only a step towards the ideal, which he expresses thus:—Provision for the insane ought in future to consist in small asylums for the treatment of acute and dangerous cases, and large agricultural colonies, with freedom and family life, for all other classes.

To those—and they are probably not a few—whose ideas of the Belgian asylum system have been gained chiefly from what they have heard of the much lauded colonies of Gheel and Lierneux, Crocq's paper will be a genuine surprise. This communication is referred to by Ferrari as giving "a lamentable picture of the private asylums of Belgium from the medical point of view." It is not to be thought, however, that Crocq brings grave charges against his professional brethren; what he attacks is merely the system that prevails, the chief blot upon which is that it permits pauper patients to be placed in private asylums.



His case rests mainly on the fact that the existing system is in this and other particulars at variance with the practice of other countries. In proof of this contention he gives details that he has collected regarding the methods of provision for the insane in eighteen other countries. He then compares with these "the medical organisation of the asylums in Belgium." It appears that there are two State asylums, twenty-two *asiles provisoires et de passage*, dependent upon the communal authority, and thirty-seven private asylums. By far the greater number of the insane are placed in these private asylums, about half of which receive pauper patients. Some of them are for the accommodation of such patients only. In 1897, the private asylums contained 71·5 per cent. of the insane, the public asylums 28·5 per cent. The corresponding figures in England are 3 per cent. and 97 per cent. Certain grievances relating to the position, number and remuneration of the medical officers are also dealt with. The paper is, in brief, a strong appeal for the reconstruction of the Belgian asylum system in accordance with the best models presented by other countries.

Dr Ferrari, after explaining the origin of the Congress (which was presided over by Dr Peeters of Gheel), enumerates the subjects that were set down for discussion. These were (1) the present position and value of the system of family care of the insane and that of care in asylums, from the scientific, humanitarian and economic points of view; (2) categories of the insane for whom family care and treatment in asylums are respectively the better suited; (3) the scientific organisation and administration of colonies and asylums; (4) the need of the establishment of additional family colonies for the insane and rules therefor. Only the first of these questions was fully discussed. The family colony system, which was seen in operation at Gheel and Lierneux, produced a most favourable impression on the Congress. Dr Ferrari, referring to this subject, expresses the opinion that the family colony with its central infirmary, though it will not destroy the existing asylums, since it cannot suit all cases of insanity, will henceforth constitute the ideal form of provision for the insane, and that it will moreover serve in a practical, active and economic way, to arrest the otherwise fatal enlargement of the central asylums.

Representatives of various nations, chiefly the official delegates of their governments, made formal statements regarding the care of the insane in their respective countries. From these statements the following general facts could be gathered:—(1) Insanity is everywhere undergoing general and progressive increase; (2) everywhere the existing asylums are overcrowded, and in many countries they are continuing to construct new asylums at great expense; (3) the difficulty of providing for the increased number of the insane cannot be satisfactorily met by

making use of work-houses and hospices ; (4) in many countries resort has been had to the system of family care which has everywhere proved very successful ; (5) in the countries that are most advanced the medical director is generally given supreme authority over all the services of the asylum ; (6) the supervision of the care of the insane is in most countries intrusted to competent central inspectors ; (7) in the more advanced nations the idea prevails that greater liberty should be granted to the insane, and that their admission and discharge should be facilitated, so that the asylums may be given the character of ordinary places of treatment ; (8) in the best regulated asylums all methods of coercion have been abolished.

After giving an account of the extent to which the system of family care is at present adopted in various countries, Ferrari deals with the papers that were read before the Congress, referring specially to that of Crocq, which seems to have given rise to a very heated discussion. He then summarises the resolutions adopted, which are given in detail at the end of his report.

A point about which there was considerable difference of opinion was the exact form that the system of family care ought to take. Some were in favour of free colonies like those of Gheel and Lierneux. Others advocated the attached village system of Tamburini. Another section preferred the German plan, in which groups of five or six cottages of similar type are provided for the families of asylum attendants with whom the patients board. Lastly, we are told that to the Scottish system only the English representatives remained faithful ! It was, however, freely recognised that fixed rules could not be laid down regarding this matter, as a system of family care that was best suited for one country, might not be adapted for another.

An entire sitting was devoted to the subject of the care of the mentally deficient. The question of the provision that ought to be made for epileptics was also touched upon. Regarding the latter, Ferrari remarks that we certainly cannot long maintain the present state of matters in which only the ordinary asylums are available for them,

The resolutions adopted by the Congress, which are characterised by Ferrari as the last word in a debate that ought to be of interest to every alienist, are as follows :—

I. Family care of the insane.

1. The Congress expresses the opinion that the system of family care, in all its forms, should be applied in the largest measure.
2. The most practicable way of initiating the system of family care is to institute it in the villages near asylums, the patients being placed with carefully



selected families, and kept under the regular and direct surveillance of the doctors of the asylum.

3. (a) For a very considerable proportion of the insane, the family colony represents the most free and least costly system of care, as well as an important curative co-efficient.
- (b) The system of family care can be added to every asylum institution that is organized in accordance with modern requirements, especially when the attendants and their families are in favourable conditions as regards their houses, etc.
- (c) The generalisation of this form of care can only be effected by putting it in practice around large establishments in small groups that can form the nucleus of origin for true family colonies.
- (d) The adoption of the system of family colonies can arrest, actively and economically, the continual encumbrance of the asylums.

#### II. Admission of patients.

Considering that easy admission and early entrance into hospital constitutes the surest guarantee of the successful treatment of insanity, it is recommended that every facility be given, both for treatment outside and for speedy admission to asylums, from the first signs of illness, and even (in cases of absolute urgency) without the necessity of a certificate.

#### III. International reciprocity in regard to *assistance*.

The Congress expresses the opinion that arrangements should be made between the various governments for reciprocity in regard to the succour to be given to the infirm in general, to the insane in particular, to deserted children, etc., each undertaking to extend succour, in their own territories, to all those who have need of it.

#### IV. Sanitary and scientific service.

- (a) The directorship of the asylum should be intrusted to the doctor, both as regards the sanitary service and the administrative.
- (b) Every asylum ought to have at least one doctor for every 100 patients. In the interests of the patients intrusted to them, the doctors ought to live in the asylum.
- (c) There ought to be instituted visiting doctors to attend the insane poor, brought to the notice of the authorities and to be treated in their own homes, in hospitals, or by family care.
- (d) All asylums ought to be furnished with the scientific laboratories necessary for the study of all that can con-

tribute to the diagnosis of disease, and to the progress of mental medicine.

V. Coercive measures and the employment of patients.

Opinions were expressed in favour of the abolition of measures of coercion, and in support of the work of the patients being always carried out under the direction of the medical officers, who should prescribe its nature and duration.

VI. Attendants.

(a) The condition of attendants in asylums should be ameliorated as far as possible, and their future, together with that of their families, should be made secure.

(b) Theoretical and practical professional instruction should be given to the attendants by the medical officers of the asylum, who should also test its results.

VII. The mentally deficient.

1. There ought to be created in the communal schools special classes for deficient, under medical supervision.

2 The Congress is of opinion—

(a) That medico-instructional institutions for deficient should be very generally developed and increased in number.

(b) That instruction should be not only intellectual and moral but also practical, technical and manual, and such as to fit for a useful occupation.

(c) That committees of guardianship should be instituted for the weak-minded who have passed through these institutions, with a view to having them still watched and helped.

(d) That the management of these medico-instructional institutions should always be intrusted to a doctor.

(e) That abnormal school children should be placed in educational asylums.

(f) That there should be instituted courses of scientific teaching applied to the treatment of the mentally deficient.

3 That a study should be made of the conditions of a social nature that lead to congenital weak-mindedness, that is to say those that are capable of exerting an influence upon the mother during gestation, parturition, etc., and those that consist in diseases of early infancy, and that a campaign should be initiated with the object of devising practical measures by which to prevent and combat them.

W. FORD ROBERTSON.

## Proceedings of Societies

### TREATMENT OF EPILEPSY.

At a meeting of the Medical Society of London, on February 9th, 1903, a discussion on the treatment of epilepsy was opened by Dr Risien Russell and Sir Victor Horsley.

Dr Risien Russell, in opening the discussion, said that his remarks would be confined to the subject of idiopathic epilepsy, and that it would be well to consider: (1) Whether any researches have supplied evidence that can be regarded as positive or reasonable proof of the etiology and pathology of idiopathic epilepsy. (2) If so, whether any line of treatment has been established on the sound basis of this knowledge. (3) If not, whether any treatment has been introduced which, while not attaining to this highest ideal, has nevertheless supplanted or supplemented the treatment by bromides. (4) If no such treatment has been evolved we may finally consider how the bromide treatment can be conducted with the greatest advantage and least disadvantage.

Dr Russell reviewed the various theories which have been put forward to explain the origin of epilepsy, and stated that the theory of auto-intoxication has the most numerous supporters; he did not consider that the question has yet been answered in such a way that the treatment of the disease could be put on a sound etiological or pathological basis. Reference was made to the hitherto unsatisfactory results which have been obtained from an attempted serum-therapy and from the inoculation of patients with filtered cultures of certain micro-organisms. The conclusion was arrived at that the bromides are still our most potent agents in the combating of epilepsy, and the author expressed a strong opinion that these salts do not deserve the bad reputation which they have gained in the public mind, and that their supposed ill effects were nearly always due to the disease rather than its remedy. He did not think that the bromides of sodium and ammonium were to be preferred, except as a change, to that of potassium, but he suggested that the bromide of camphor merited further trial in view of the results obtained by Bourneville. Much stress was laid on the importance which attends the proper administration of bromides in such a way that the maximal effect is produced with minimal doses, and on the importance of correcting errors in diet, such as the excessive use of tea, coffee, and meat. Useful adjuncts to the bromides were to be found in borax, arsenic, digitalis, belladonna, etc., but these drugs could not be said to have any valuable influence on the attacks when prescribed by themselves

Of the more recent compounds which have been introduced, bromopin has found most favour. The most important modification of the dietetic treatment of epilepsy was the exclusion of ordinary salt from the food of the patient, a step which had been foreshadowed many years ago by Dr Hughlings Jackson, and which was based on the supposition that the bromide salts effect their results by replacing the chlorides in the tissues. In conclusion, Dr Russell pointed out how much can be done to alleviate the condition of epileptics by placing them under more favourable circumstances, such as can be found in epileptic colonies.

Sir Victor Horsley, in introducing the subject of the surgical treatment of epilepsy, remarked that epilepsy should be regarded as a symptom rather than as a disease. He had had no experience of the surgical treatment of generalised idiopathic epilepsy, but had operated on five cases of focal idiopathic epilepsy. In two of these he had found gross lesions, one of syphilitic and one of tubercular nature; in two others he had found no lesion. In the fifth patient, whose spasms were limited to the left facial region he exposed the foot of the right ascending frontal gyrus, and found that part congested; the removal of the focus of congestion was followed by the establishment of general idiopathic fits, except in the left facial region. Sir Victor Horsley classified cases of Jacksonian epilepsy as either traumatic, congenital, neoplastic or reflex in origin. When general convulsions followed a severe blow on the head, without obvious evidence of injury, surgical measures were generally of no avail. On the other hand, in the case of a definite trauma of the skull followed by localised convulsions, perhaps becoming general later, the question of successful surgical intervention depended largely on the situation of the injury; as a result of experience it was found that trephining in the frontal region was unfavourable, and in the occipito-temporal and occipito-parietal generally, but not always, unattended by benefit. When the skull injury was over the motor cortex trephining should always be carried out and the scar and circumferential tissue removed. In cysts and porencephaly of congenital origin operation in children was often beneficial. The neoplastic cases were to be judged on their individual merits as fits under these circumstances depended on many various factors, and were not in themselves sufficient evidence to guide the surgeon in regard to operative measures.

The speaker had performed a laminectomy on two cases in which fits had followed injury to the spine, and in one of these the operation had been followed by the cessation of all attacks, while in the other no beneficial results had accrued. In conclusion, hystero-epilepsy offered no field for surgical interference.

Dr Beevor argued against the toxic origin of epilepsy and

corroborated the opinion expressed by Dr Russell on the efficiency of the bromides and the paucity of ill effects attending their use. Paraldehyde had been successful in one case where bromides had failed. The use of opium was certainly to be deprecated.

Dr J. A. Ormerod thought it was essential to watch cases of epilepsy for long periods of time ; and by long periods he meant many years, not months. He considered that borax was useful in cases which had been taking bromides for a long time, and that belladonna was often helpful in petit mal. Those patients who failed to respond to bromide treatment should be made the subject of very careful observation, whilst more study might be given to the influence exerted by exciting causes of the attacks, such as digestive disturbances, etc.

After Dr Tuke had referred to some curious features displayed by epileptics who were also insane,

Dr Turner endeavoured to show by means of statistics, that equally good results had been obtained in the treatment of epilepsy before the introduction of bromides. Comparisons were hardly permissible, however, on account of the absence of unanimity as to what constitutes a cure.

Dr Langdon Down had not found potassium bromide to be efficacious in cases of minor epilepsy, and had sometimes thought the use of that drug was actually harmful in those patients. He referred to the effect produced by an epidemic of influenza and pneumonia among a number of epileptics ; although the attacks had been temporarily arrested, they had returned afterwards in so virulent a form that two patients had died in statu epileptico.

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## Review

**DIE AKUTEN GEISTESKRANKHEITEN DER GEWOHNHEITSTRINKER.** Ein klinische Studie von Dr K. BONHOEFFER, Privatdocent in Breslau. Pp. 226. Gustav Fischer in Jena, 1901. 5 M.

THIS work embodies the results of many years' experience in the Psychiatric clinic and in connection with the prison in Breslau. It is not here possible to do more than indicate a few of the author's conclusions.

He believes that much has hitherto been included under the category of alcoholic psychosis which does not belong to it. He treats the subject under four main heads:—1. *Delirium Tremens*—Visual preponderate over auditory hallucinations, the converse of

what obtains in acute hallucinatory insanity of alcoholics; the characteristic thing however being not this, but the combined occurrence of hallucinations referable to different senses. As to Rose's view that visions of animals seldom occur of themselves, but depend on suggestion, it was found that though their frequency has been over-estimated, they do occur relatively often. The rapidly changing visual sensations consequent on the bodily movements occurring in the disease, may suggest animal life. While præcordial anxiety is frequent, "hypochondriacal" feelings referable to the abdominal viscera, do not arise from alcoholism alone.

Experiments conducted in cases free of neuritis, indicated that analgesia when present is not peripheral but psychic. Hearing and smell remain unaffected. Magnan's statement that amblyopia occurs as part of D. T. was not confirmed; a result of chronic alcoholism having been attributed by that author to the intercurrent delirium. Though loss of colour perception was not found, there seemed sometimes to be a blunting of this sense. Out of 30 cases examined with special care, only 3 showed a contraction of the field for white, and that only to the extent of cm. 5 and 10 degree, a result which does not tally with Kruckenberg's observations. In some cases after the delirium a transitory narrowing for blue and red was discovered.

The maximum degree of attention attainable under special stimulus for a short period may nearly reach the normal standard; but the average of sustained attention falls far short of this. That this impairment occurs in stages when hallucinations are not present, points to there being a primary affection of the faculty, and not merely disturbance caused by hallucinations. In investigating the power of apprehension, it was found that simple pictures were usually, though not always, understood. Cases occurred where an incorrect answer depended on an "inner" and not on an "outer" resemblance. The influence of suggestion is more pronounced here than in any other acute psychosis. Touching an object which has been misinterpreted when merely seen, often dispels error; more rarely the converse obtains. Attempts to produce hallucinations of taste and smell by suggestion were not successful; and as to the contents of memory, suggestibility holds only for the recent, not for the remote past. Sense of personality unlike that of time and place, remains intact.

The author had the exceptional opportunity of performing post-mortem examinations two hours after death. On no occasion was cedema of the pia found. In many cases there was neither marked anæmia nor marked hyperæmia of the brain, though any departure from normal tended to be in the direction of the latter. Sections were stained by Golgi's, Marchi's, and Weigert's methods. In two cases of very severe delirium, marked degenerative changes



(Marchi's method) were found corresponding with the entering fibres in the radial fibrillation of the central convolutions, while in sensory parts in the temporal and occipital regions no broken-down medullary layers were seen. Disintegration of the medullary sheath as it is found in D. T. has certain seats of election in the brain, in the neighbourhood of the central convolutions and the vermis of the cerebellum. There is a marked tendency to hæmorrhages, the favourite seats being the grey matter round the third ventricle and aqueduct and the region above the nuclei of the eye-muscle nerves; also within that of the nucleus for the oculomotorius and down as far as the abducens nucleus. In the spinal cord, disintegration was found to be most marked in the posterior tracts, especially in Goll.

No case of D. T. was met with in persons whose drink was confined to beer and wine.

The author believes (see also his later article in *Berlin. kl. Woch.* for 1901, No. 32) that in seeking a theory of the pathogenesis of the disease, neither the view of its being a mere exacerbation of a chronic process, nor the so-called "inanition" explanation are sufficient. Besides chronic alcoholism there must be the flooding of the system with toxic substances not identical with alcohol.

2. *Chronic Alcoholic Delirium or Korsakow's Disease.*—The onset is usually in a delirious, but may be in a stuporous condition, and may even begin with gradual loss of memory. The occurrence of a sleep apparently critical, but not followed by corresponding mental improvement may justify the suspicion that the case is other than D. T. Symptoms of an asymbolic nature, or even paraphasia may be present. The impairment occurring in the higher sensory realms is first noticed in the auditory. One patient could not repeat unknown words after 1-2 min.; pictures (the mental image of which is normally more permanent) could not be recalled in 1-1½ min. It may be found that a picture is shown several times without obvious result, yet later the real impression it has produced appears unexpectedly in a future answer the patient makes. In many cases "retroactive amnesia" is present, the memory of events of the highest personal importance being blotted out. The point where memory is lost may be sharply defined, though not always so sharply as appears. Memories at the borderland are excessively clear, so that for the time they exist more as actually present situations than as mere memories. What the author only once saw in D. T.—in a case which, however, did not have a clear crisis—viz., that the occupation delirium related to a more remote past, an employment in which the patient was engaged a year previously, is here the rule. In testing the power of apprehension pictures were shown, and the reaction—time

before the patient answered as to their meaning noted. This was found to be longer than in health. The neuritis symptoms which are seldom absent generally precede the mental both in onset and in disappearance. Delay in perception of pain was very marked in three cases. Two patients died of trophic lesions, pemphigus-like in form, and not confined to pressure areas. Affections of the vagus are presumably pretty frequent. A series of seven cases showed a remarkable cyanosis, tachycardia, and dyspnoea, together with "short breath" speech. Such symptoms may disappear along with those of neuritis elsewhere. Disturbances of swallowing and tongue movements, as well as paralysis of the soft palate were observed, but not peripheral flaccid palsy. Frequently an acute disturbance of exterior eye-muscles, especially the abducens, and more rarely ptosis is met with. *Polioencephalitis hæmorrhagica superior* (Wernicke) cannot be regarded as a disease *sui generis*. There occur in the acute stages of Korsakow's disease multiple hæmorrhages, especially in the neighbourhoods of the thalamus and aqueduct and of the nuclei of the motor nerves of the eyes as far as the fourth ventricle. If these be emphasised in the latter region the picture is that of polioencephalitis hæmorrhagica superior. Hæmorrhages occur also in the cerebral cortex, the primary cerebral peduncle, cord, and peripheral nerves. In a case of severe Korsakow's disease with extensive neuritis in the extremities and of the cranial nerves, death occurred with the development of a large hæmorrhagic softening in the cerebellum. The symmetrical occurrence of hæmorrhages in places supplied by end arteries might suggest their dependence on thromboses due to toxic substances in the blood. In all cases of chronic delirium there are changes in the cord, especially in Goll's tract. In two cases of extensive alcoholic neuritis a degeneration was found spreading out in butterfly form in the posterior columns; a narrow degeneration lay to both sides of the middle line in Goll's tract, the outer part of which was free. A second zone extended wing-like from the anterior end of Burdach's column sideways and backwards.

3. *Acute Alcoholic Hallucinatory Insanity* ("Acute Hallucinosé der Trinker"), and 4. *Transitory disturbances of consciousness of chronic alcoholics* are next dealt with in detail, but space does not permit of their consideration here.

With a view to finding an hypothesis which would explain why one person develops D. T. and another alcoholic hallucinatory insanity, use is made of suggestions of Charcot and Wernicke as to individual differences in the form of thinking—the thought of some persons being carried on chiefly in words, that of others in memories and picture images. Now the irritation symptoms in D. T. are chiefly to be referred to the areas of visual and tactile perception, while in A. H. I. one must recognise a marked central



hyperexcitability of the hearing and word hearing areas. The latter disease affects chiefly those whose work is of a mental character.

One must admire the persevering and critical spirit as well as the ingenuity which this book displays. The author takes note of transitional forms and has avoided the unfortunate error of extreme subdivision. Special difficulties are met with in the study of the diseases in question; and it frequently happens that the most important cases from a scientific point of view are just those where detailed clinical examination is practically impossible, while for the framing of statistics the "histories" are in many cases misleading. Whether all the author's methods and conclusions can be looked on as final, it would perhaps be premature to say; but if those who have opportunities of studying these diseases did so on similar or analogous lines, in the course of time individual errors would be eliminated and the subject placed on a surer foundation. The desirability of early post-mortem examination, especially for higher cytological work, is self-evident, and the fact that we have here records of such where section was performed two hours after death, adds an important element to the value of Dr Bonhoeffer's monograph.

A. HILL BUCHAN.

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## Original Articles

### **BACTERIOLOGICAL INVESTIGATIONS INTO THE PATHOLOGY OF GENERAL PARALYSIS OF THE INSANE.**

#### **Preliminary Note.**

By W. FORD ROBERTSON, M.D., G. DOUGLAS M'RAE, M.B., C.M.,  
and JOHN JEFFREY, M.B., F.R.C.S.Ed.

IN continuation of previous investigations\* which seemed to show that the toxæmia of general paralysis of the insane is of gastro-intestinal and bacterial origin, and that the part played by syphilis, alcohol, lead, etc., in the pathogenesis of this disease is essentially that of altering the natural immunity, we began more than a year ago, at the Royal Edinburgh Asylum, a research with the object of ascertaining if cases of general paralysis have any distinctive features in the bacterial flora of the alimentary tract, and if micro-organisms are commonly present in the blood and internal organs.

In a series of cases we made cultures from the blood during life, but the media remained uniformly sterile, and we therefore abandoned this line of investigation.

Obtaining post-mortem examinations under as favourable conditions as possible, we have made cultures from the wall of various parts of the alimentary tract, from the gastro-intestinal contents, blood, and various internal organs. One of us (W. F. R.) has also pursued the enquiry by histological methods in the same cases, and also in others.

\* Lewis C. Bruce, *Brit. Med. Journ.*, 29th June 1901; W. Ford Robertson, do.

We hope to publish shortly a full account of the results of these investigations, and to discuss their bearing upon the question of the pathogenesis of general paralysis of the insane. In the meantime we wish merely to record two outstanding facts that have been elicited. First, there is evidence that not only the alimentary canal, but also the respiratory tract, is the seat of origin of a severe toxic infection in general paralysis. The second fact is, that whilst the causal agents of this toxic infection from these regions are represented by various bacterial forms, there is one micro-organism which appears to have a special significance. It does so by reason of the seeming constancy of its presence, the enormous numbers in which it can be shown to occur in some cases, the frequency with which it takes part in a terminal general invasion, the known pathogenic characters of the group to which it would seem to belong, and the comparative rarity with which a similar organism can be isolated from other cases when the same technique is employed. This organism is one that resembles the Klebs-Löffler bacillus. It rapidly produces acid in glucose-broth, and is therefore not Hofmann's bacillus. It is most readily obtained from the mucous surface of the stomach, from the tonsils and bronchi. Its separation from the other organisms present is naturally a matter of considerable difficulty, but can generally be accomplished by making sub-cultures from the inoculated medium within six or nine hours, as was suggested to us by Dr Shennan. Pure colonies can sometimes be obtained in this way in the first sub-culture. Ordinary culture media, but especially blood-serum, are suitable for growing the organism. We have, however, chiefly used a special medium, which we find can be depended upon to show the bacillus in the form in which its metachromatic granules are prominent, and in which, therefore, it is distinguishable morphologically from organisms that do not belong to the diphtheria group.

Facts of the kind we have mentioned do not, of course, afford proof that this organism plays any essential part in the causation of general paralysis. They seemed to us, however, to warrant a careful experimental enquiry into its virulence and pathogenic action. This enquiry has kindly been undertaken for us by Dr Shennan.



**EXPERIMENTAL OBSERVATIONS UPON THE PATHOGENIC ACTION OF AN ORGANISM RESEMBLING THE KLEBS-LÖFFLER BACILLUS, ISOLATED FROM CASES OF GENERAL PARALYSIS OF THE INSANE.**

**Preliminary Note.**

By W. FORD ROBERTSON, M.D., and THEODORE SHENNAN, M.D.\*

WE have used pure cultures of the organism resembling the Klebs-Löffler bacillus, isolated from various cases of general paralysis, in the course of the investigations referred to in the preceding note.

Intra-peritoneal and subcutaneous injections in guinea-pigs have not produced any noteworthy morbid symptoms. Intra-pleural injection in a white rat resulted in death of the animal in five days. Microscopic examination of the tissues in this case showed that the organism had multiplied at the seat of injection and had spread into the adjacent pulmonary tissues and also into the pericardium. Three rats were fed for several weeks upon bread mixed with unsterilised broth cultures of the organism. For the first three or four weeks the animals seemed to remain well. After this time, however, they began to show morbid symptoms, which gradually increased in severity until the rats became acutely ill. At first they showed especially slowness and uncertainty of gait and drowsiness after feeding. Later, they manifested distinct motor weakness, marked inco-ordination of movement, dyspnoea, great drowsiness, and looseness of their motions. One was killed with chloroform when it appeared to be moribund. In the case of the other two, which were the subjects of a later experiment, the disease was allowed to go on to a fatal termination. In all three cases the morbid phenomena exhibited during life were of the same character. Control animals kept under identical conditions remained healthy. Microscopic examination of the tissues has revealed a series of pathological changes, differing in the individual cases only in degree. In the non-nervous organs the alterations consist especially of gastro-intestinal catarrh (localised particularly in the upper part of the

\* The experiments have been made by Dr Shennan.

small intestine), and proliferative and degenerative changes in the liver. There is also evidence of inflammatory changes in the lungs. The brain shows severe degeneration of a large proportion of the cortical nerve cells, early acute periarteritis, proliferation of the neuroglia, especially in the first layer of the cortex, proliferation of mesoglia cells, and infiltration of the pia-arachnoid. The cord shows similar changes, but the degeneration of the nerve cells is more advanced. Many of the nerve fibres exhibit distinct morbid alterations, both in Marchi and in Weigert-Pal preparations.

Whatever the relation of this organism may be to general paralysis of the insane, the evidence we have obtained distinctly shows: (1) that when introduced by way of the alimentary tract in the form of broth cultures, it is capable of producing in the rat a series of morbid phenomena which especially affect the nervous system, and which, when once established, may go on progressively till death results, even though feeding with the cultures is stopped; and (2) that the associated changes in the central nervous system have a distinct resemblance to those which occur in dementia paralytica.

We propose to publish shortly a full account of these and other experimental observations with this organism, including a description of the morbid changes that we have found to be produced in the tissues.

## **THE DESIGNATION OF MUSICAL NOTES IN SCIENCE AND MEDICINE.**

By SIR W. R. GOWERS, M.D., F.R.S.

THE convenient and accurate designation of musical notes has become a subject of importance to medicine and to science, especially to physiology. The system current among musicians has come down from the early days of the organ,\* but it was adopted by Helmholtz and has therefore been used by most writers since the publication of his classical work on "Tönempfindungen." But it is a system which has no rational foundation,

\* It originated in the sixteenth century by a development of an earlier system, due to the extension of the notes of the organ. See Rockstro's Art. "Tablature," Grove's "Dict. of Music."

except in organ construction, and with this it has only a partial connection. The absence of intelligible ground for it has had the necessary consequence of mistakes by those who have attempted to apply it to wider needs. It is not too much to say that error and confusion is met with as frequently as precision.

The need for precise designation occurs in describing normal hearing, and still more in recording the limitations of hearing met with in disease. The latter is as frequent in the work of the physician as in that of the aural surgeon. In the various senile and gouty changes in the internal ear, so often associated with vertigo, in the results of other forms of ear disease and central changes, and in the atrophy of the auditory nerve sometimes met with in tabes, there is often peculiar restriction of the range of audition. The method of designation by means of the number of vibrations per second is not convenient except for the higher notes, above the range of the tuning fork. For lower notes the musician's method of designating the notes by letters and distinguishing the successive octaves is far better, and is generally employed.

The term "octave" is in universal use as a name for each series of seven notes. It is apparently due to the fact that the eighth note is in unison with the first, although it really belongs to the next "octave." The more accurate term "septave" has been occasionally employed, first nearly a hundred years ago,\* but few musicians have ever heard it. Since the series of musical notes, without added sharps or flats, has C for the key-note, the system of making each designated octave begin with C is universal. Each note of the series of seven notes being indicated by a letter, the successive series or "octaves" are distinguished by a number, or by strokes, after each letter, above for the higher, below for the lower. Instead of strokes or numbers, the method of doubling or trebling the letters has also been employed.

To distinguish the higher from the lower, some starting-place is necessary. The great source of confusion is that, in the current system (or what is supposed to be such) the starting-place for the higher and lower octaves is not a singly conveniently placed unnumbered octave, but is constituted by two octaves,

\* In a rare little book on "The Art of Tuning," by Earl Stanhope, Lond. 1806. A copy is in the Library of the Athenæum Club, London.

both unnumbered, inconveniently placed, and distinguished by a difference in the letters that indicate the notes. The upper one is indicated by small letters, the lower by capital letters. Each is unmarked, but the former is called the unmarked octave (the "unstroked," "ungestrichen" octave, because strokes were used of old instead of letters); the latter is distinguished as the great octave, apparently from some relation to the "great organ." The "unmarked" octave extends up from the C in the middle of the bass, the great octave from the C on the second leger-line below the bass.

This method will be better understood if the designations are added to the note-symbols as in the following table. It must be remembered that each octave extends upwards from the C to the B above. I have added to it a serial indication of some of the errors which are to be found in scientific and medical writings.

C in altmo.  
Contra C. Great O. C tenor. C middle. C treble. C in alt.

C = 33    66    132    264    528    1056    2112    4224

	Contra.	Great.	Un- marked.	One time marked.	Two times marked.	Three times marked.	Four times marked.	Five times marked.
1. Helmholtz	C <sub>1</sub>	C	c	c <sup>1</sup> or c'	c <sup>2</sup> or c''	c <sup>3</sup>	c <sup>4</sup>	c <sup>5</sup>
2. Tyndall	C <sup>1</sup>	C	c	c <sup>1</sup>	c <sup>II</sup>	c <sup>III</sup>	c <sup>IV</sup>	c <sup>V</sup>
3. Bosanquet	C or C	C	C <sup>0</sup>	C <sup>1</sup>	C <sup>2</sup>	C <sup>3</sup>	C <sup>4</sup>	C <sup>5</sup>
4. Banister	CC	C	C	c	c	c	c	c
5. Peterson		C <sub>1</sub>	C	c	c'	c''	c'''	c''''
6. "Acoustics" Encyc. Brit.				C	C <sub>1</sub>	C <sub>2</sub>	C <sub>3</sub>	
7. Foster	C <sub>1</sub>	C	C	C <sup>1</sup>	C <sup>2</sup>	C <sup>3</sup>		
8. M'Kendrick	Do <sub>1</sub>	Do <sup>1</sup>	Do <sup>2</sup>	Do <sup>3</sup>	Do <sup>4</sup>	Do <sup>5</sup>	Do <sup>6</sup>	Do <sup>7</sup>

In Politzer's "Handbook of Diseases of the Ear," Eng. trans., Cassells, 1883, the same note, C = 512, is referred to on p. 167 as C<sup>3</sup>, and on p. 178 as C̄.

1. Helmholtz, "Tönempfindungen," dritte Ausgabe, 1870.
2. "Lectures on Sound," quoting from Helmholtz.
3. Encyc. Brit., art. "Music," part ii. The capitals for the

upper notes are about one quarter smaller than those for the lower.

4. "Handbook of Music." The double C for C<sub>1</sub> is open to mistake, although really clear.
5. Art. "Harmony," Chambers's Encyc.
6. Author's initials only given. The actual note adduced is A. "A<sub>3</sub> is three octaves above the A between the second and third lines of the treble clef."
7. Sir M. Foster's "Manual of Physiology," all editions. Only the upper and lower notes are given, but the series follows from the vibrations assigned to them.
8. "Physiology of the Senses." "Fa" is the note given, but from the vibrations assigned to it in the several positions, the above series follows with no unmarked octave.

More examples might be adduced, but these suffice to show the confusion that has resulted under the present system of two unmarked octaves, in an arbitrary situation, for which no clear reason exists to help the memory or to induce uniformity. The distinction of capitals and small letters, the former for the few octaves below the C within the bass, and the latter for it and the numerous octaves above it, has evidently been felt by some writers to be an anomaly not worth conformity. This seems to have been the opinion of Grove, since in every article in his "Dictionary of Music" similar capitals are employed for the high and low notes, with the distinction of some descriptive designation.

It is not worth while to discuss in detail the examples of variation I have given. Most are simple mistakes, as their examination will show. I have taken pains to ensure precision in every example. It is certain that in Science a more simple and a rational system is needed, if uniformity and exactness are to be secured. It seems also that in Music the need is scarcely less. There is no sharp division between Music and Acoustics, or between the latter and Physiology.

For a useful system it is essential that there should be only one unmarked octave, or "neutral octave," as I think it might be conveniently termed. The best position for this is certainly the octave above the "unmarked octave" of Helmholtz and the Germans. This position is assumed in the article on "Acoustics"

in the *Encyc. Brit.*, and, for the higher unmarked octave, by Peterson ("Harmony") in *Chambers's Encyclopædia*. Its advantages have been strongly urged by Dr Shinn, of the Guildhall School of Music, in a letter to the *Musical News*, January 24th, 1903, in the pages of which I had endeavoured to evoke some discussion of the subject. I first suggested that the higher of the two present "neutral octaves" would be most likely to obtain adoption, but the advantage of the octave above this is manifest. The neutral C is then that which is often fitly called the "middle C," between the bass and treble staves;  $C^1$  and  $C_1$  are each within the treble and bass staves respectively;  $C^2$  and  $C_2$  are each on the second leger-line, below and above the respective bass and treble, and may be called the sub-bass and supra-treble. These words are a convenient and sufficient distinction in oral description. It is not only desirable but necessary, to secure precision and accuracy, that a consistent and reasonable method should be employed.

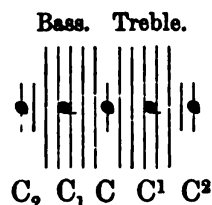
The following will then be the method that is suggested:—

33	66	132	264	528	1056	2112	4224	8448	16896
Sub-bass	Bass	Middle	Treble	Supra-					
				treble					
$C_3$	$C_2$	$C_1$	$C$	$C^1$	$C^2$	$C^3$	$C^4$	$C^5$	$C^6$

Tuning forks avail us up to  $C^3$ , but the range of hearing extends, in young persons, at least three octaves higher, and for these upper notes Galton's whistle is available. It is an instrument of the greatest service in the estimation of the function of the auditory nerve.\*

The symmetry of the suggested method will be more clearly perceived if the staves are placed vertically.

\* It is less known than it deserves to be. It is made by Hawksley, at the suggestion of Mr Francis Galton. It may be well to mention to those who use it that it should be blown through from the mouth, not breathed through from the lungs, to prevent the interior of the fine steel tube becoming rusted. For the same reason it is well to draw air through the tube after use.



Moreover, the system aids the memory of the number of vibrations of these several notes. The bass  $C_2$  is 33 vibrations. The first (leger-line)  $C$  below the bass ( $C_2$ ) is the first with two figures (66), and the first above the treble ( $C^2$ ) is the first with four figures (1056), while the number of vibrations of  $C^4$  (4224) is almost as easily remembered as  $C_2$ . This may be a trifle, but it is a convenient trifle, and I have yet to learn that even that justification can be urged in favour of the old method, venerable as it is, and familiar as it is to many.

I may be permitted to end this note with one example of the need for uniform precision. In a book, published in 1885, I wrote this: "The patient is absolutely deaf to the loudest musical notes above  $E$  of the treble clef and below the lower  $G$  of the bass." The range of hearing afterwards lessened, "until only the notes between the two  $E$ 's of the treble clef can be perceived." In a German translation\* the passage is rendered: "für die lautesten musikalischen Noten über  $E$  und unter contra- $G$  völlig taub ist." . . . "Nur noch die zwischen  $\underline{E}$  und  $\underline{E}$  legenden Noten wahrgenommen werden." I may say that the  $G$  I intended to indicate was that on the lowest line of the bass clef, an octave above the "contra- $G$ ."

### NOTE ON A VARIATION IN THE COURSE OF THE PYRAMIDAL FIBRES.

By EDWIN MATTHEW, M.A., M.B., and DAVID WATERSTON,  
M.D., F.R.C.S.E.

IN the course of examination of the spinal cord of a seven months' foetus, we noticed a condition of the pyramidal tracts in the spinal cord which deserves to be put on record.

It has been recognised that variations occur in the relative size

\* "Gehirnkrankheiten," von W. R. Gowers, übersetzt von J. Mommsen, 1886.

of the direct and crossed pyramidal tracts, but no similar case has hitherto been described, and as we have traced the variation up to the medulla, we are able to give an explanation of its origin there.

Typically in each half of the spinal cord there is present a fasciculus cerebro spinalis lateralis and a fasciculus cerebro spinalis ventralis. Each lateral pyramidal tract contains not only and principally fibres from the opposite hemisphere, but also a certain number of fibres from the hemisphere of the same side. The ventral pyramidal tract consists of fibres from the same side, which probably ultimately cross to reach the cells of the opposite ventral horn of grey matter. It is recognised that the number of fibres in the ventral tract of one side when compared both with the number of fibres in the other, and with the number in the crossed pyramidal tract, varies within considerable limits.

Using his embryological method, Flechsig has shown that in the distribution of the pyramidal fibres there are various possibilities, viz. :—

(a) All the fibres may go down the crossed pyramidal tracts and no direct pyramidal tracts be present.

(b) There may be a direct pyramidal tract on one side of the cord and not on the other.

(c) There may be a direct pyramidal tract on both sides, but the number of fibres in each varies considerably.

The variations in the distribution of the pyramidal fibres in the cord are thus of the direct pyramidal tract, the lateral pyramidal tracts being fairly constant in size and position. In our specimen the variation involved both the crossed and direct pyramidal tracts.

#### MICROSCOPIC APPEARANCES.

*Distribution of the Pyramidal Fibres.*—The transverse section of the cord in the upper cervical region shows marked asymmetry due to the almost total absence of pyramidal fibres on one side of the cord. On the other side the crossed pyramidal tract is represented by a large pale area of rather greater extent than usual, reaching a point ventral to the intermedio-lateral cornu of grey matter. The direct pyramidal tract on this side occupies a large area not only along the entire margin of the anterior medial fissure, but courses round the ventral aspect for a considerable distance, extending



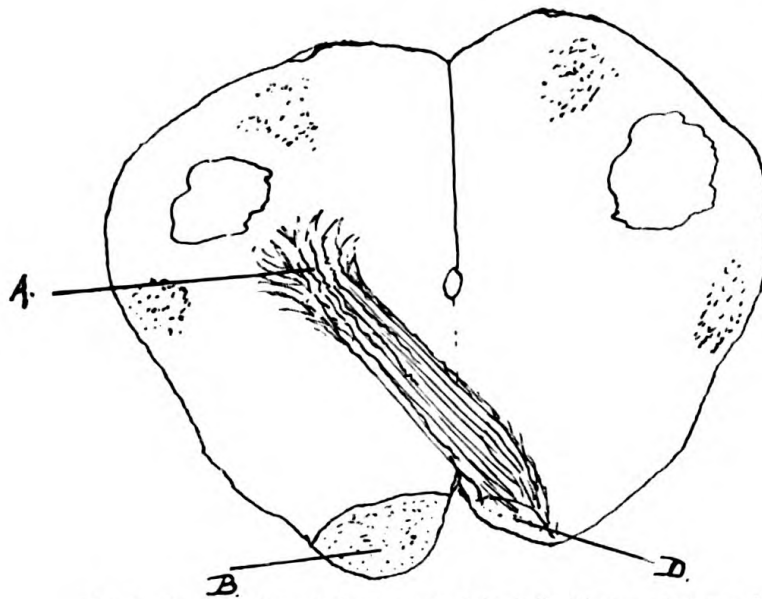


FIG. 1. Section through lower part of Medulla (*from a photograph*).



FIG. 2. Section from third Cervical Segment.

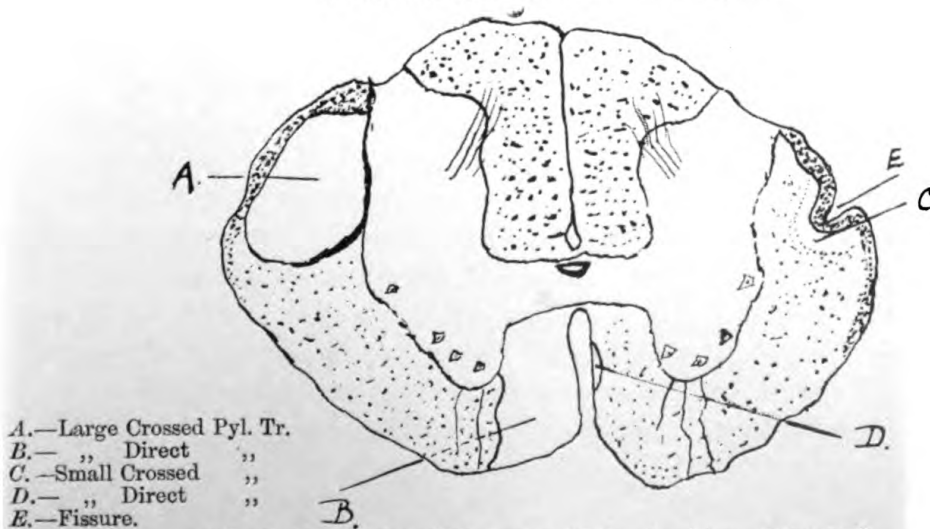


FIG. 3. Section through lower cervical region (*from photograph*).



outwards to the innermost ventral nerve root, forming an L shaped area (fig. 2).

On the other side the direct pyramidal tract is represented by a narrow band opposite the middle third of the anterior mesial fissure. On this side the crossed pyramidal tract is very small, occupying a small area between the posterior horn of grey matter and the direct cerebellar tract.

*Longitudinal Extent in Cord.*—On the side on which the pyramidal fibres are deficient, the direct pyramidal tract is very small at the level of the 6th cervical segment and absent at the 7th. The area occupied by the large direct pyramidal tract shows very little or no diminution in the cervical region. In the upper dorsal the horizontal limb of the L is somewhat less, and the tract as a whole can be traced down to the upper sacral segments.

The large crossed pyramidal tract has the usual extent in the cord and can be traced to its lower end.

*Abnormal Fissure.*—In association with and possibly a result of the very small area occupied by the crossed pyramidal tract of the one side, an abnormal fissure appears in the cervical and upper dorsal regions (see fig. 3). It begins at the 2nd cervical segment and is shallow at first, but gradually becomes wide and deep, and is best marked about the 5th and 6th cervical segments. It proceeds from a wrinkling or infolding of the pia mater, and is situated on the surface opposite the position of the crossed pyramidal tract and evidently results from the deficiency of pyramidal fibres of that side.

The direct cerebellar tract, it will be seen, follows the fissure and has been pushed inwards by the infolding of the pia.

Where the pyramidal fibres came to occupy a diminished area—below the mid-dorsal region—it disappears altogether.

*Region of Medulla.*—An explanation of this variation in the distribution of the pyramidal fibres in the cord is obtained by studying transverse sections through the medulla. In the region of the inferior olive the pyramidal fibres occupy their usual ventral position and are of equal extent on the two sides.

Sections at the level of the decussation of the pyramids, however, show that crossing of fibres takes place from one side only. The pyramidal fibres on the other evidently all pass down the same side of the cord. (fig. 1).

*What the Pyramidal Tracts in the Cord Represent.*—The large

crossed pyramidal tract therefore represents principally the majority of the fibres which have crossed over from the opposite side of the medulla.

The small crossed pyramidal tract is made up of fibres which have not decussated but have passed down to the same side of the cord. Some of these non-decussating fibres go also to form the small direct pyramidal tract on the same side. The large ventral pyramidal tract is therefore composed of the fibres from the medullary pyramid of the same side which have not, as they usually do, crossed over to form the opposite crossed tract, but have passed down along the anterior median fissure of the same side.

Prof. Obersteiner has suggested that an explanation for such and other variations is to be sought in the fact that the pyramidal fibres from the cortex are among the latest in development and in medullation. When they pass down to reach the cord the other columns of white matter are already in position, and the pyramidal fibres have as it were to search out a place for themselves and distribute themselves over the two crossed and two direct pyramidal tracts.

### **LESION OF THE FIRST DORSAL NERVE ROOT.**

By EDWIN BRAMWELL, M.B., M.R.C.P.

UNIRADICULAR root lesions are of rare occurrence, and even when met with are apt to escape recognition. The following case, which presented very definite physical signs, appears for these reasons to be worthy of a short note.

J. S., a plumber, aged 18, had always enjoyed good health. He had not had rheumatism, nor had he suffered from any symptoms suggestive of lead poisoning.

He stated that for the last three or four years he had from time to time complained of pains down the inner side of the right forearm, that a year ago he began *gradually* to lose the power of his right hand, and that a month or two later it was noticed to be thinner than its fellow. The hand used to feel cold at times. During the past six months the condition of the hand had become no worse, although there had been no improvement. Apart from the weakness and cold feeling in the hand and the pains in the arm, the patient felt in perfect health.

Upon examination there was found to be distinct flattening of the thenar and hypothenar eminences on the right side; the flexor tendons where they cross the palm were unduly prominent, and there was a slight tendency to the bird-claw hand (Plate 4). All the movements of the right upper extremity, including extension and flexion of the hand at the wrist, and extension of the fingers, were as well performed as on the left side. There was marked weakness of both the deep and superficial flexors of the fingers on the right side (grasp, right = 43; left = 80), of all the intrinsic muscles of the thumb and little finger, also of the interossei and the lumbricals. The loss of power nowhere amounted to a complete paralysis. The movements of adduction and flexion of the thumb were less impaired than those of abduction and opposition. The electrical excitability of the paretic muscles were very greatly reduced, but nowhere was there a true R.D. No fibrillary tremors were to be seen.

The cold feeling in the right hand and the pains in the right forearm have been already referred to. There were no abnormal subjective sensations in the arm. Over an elongated area on the inner side of the right forearm, extending both on to its anterior and posterior aspects from two or three inches above the elbow to just above the wrist, there was very distinct impairment of sensation. The limits of this area were very well defined (Plate 4); they were examined on several separate occasions and found to be constant. Over this area light touches were not recognised, the perception of painful impressions was distinctly reduced, while heat and cold stimuli, especially the former, were often incorrectly named.

There was no ptosis, and no nystagmus; the pupils were equal, and contracted well to light, dilating readily when shaded. There was no weakness of the lower limbs or opposite arm, and no defect of sensation to be detected anywhere, with the exception of the area above referred to. The biceps, triceps, and supinator jerks were present, equal on the two sides and not increased. The knee and Achillis jerks were active and equal. There was no ankle-clonus, and plantar stimulation gave a flexor response on both sides. The action of the vesical and rectal sphincters was not interfered with. There was no pain in the back, and no spinal curvature, vertebral prominence or tenderness. There were no trophic changes in the skin, joints or nails.

The heart was not enlarged, and the sounds were pure. There was no evidence of arterial disease. The urine contained neither albumen nor other abnormal constituents. There were no signs of lead poisoning.

*Remarks.*—The local paralysis and wasting can only be dependent on damage to the lower motor neuron.

The distribution of the paralysis and of the anaesthesia cannot be accounted for by a lesion of any one or more of the peripheral nerves of the upper extremity.

There are no symptoms pointing to disease of the spinal cord. It is perhaps conceivable, but at the same time most improbable, that the symptoms may be due to a very local focus of disease in the spinal cord. Such a lesion would require to be confined to a single "segment" of the cord, to be strictly unilateral and to involve both the anterior and posterior horns, leaving at the same time uninjured the pyramidal tracts of implication of which there is no evidence.

On the other hand the symptoms suggest a lesion of the first dorsal root.

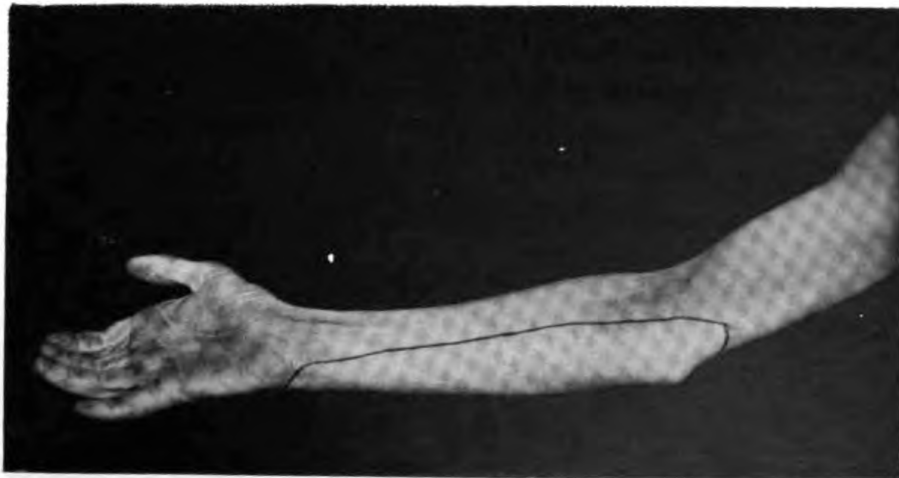
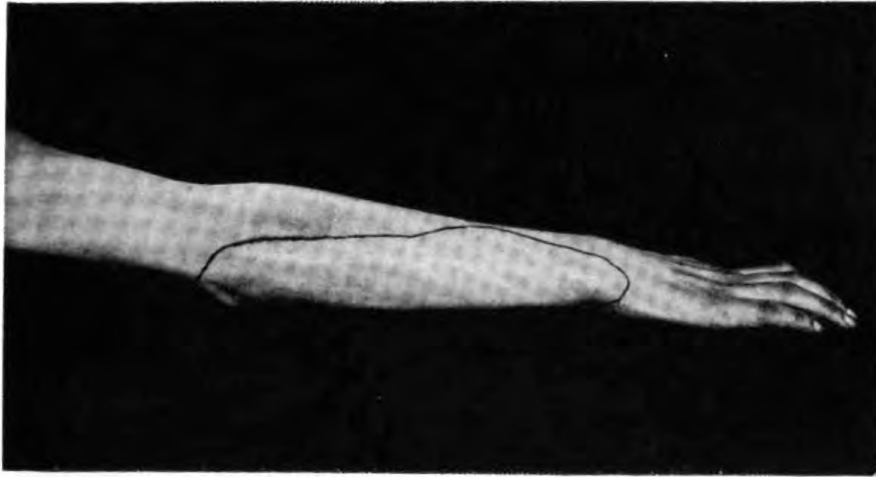
The distribution of the paralysis and of the sensory loss is almost identical with that found in three cases recently described by Farquhar Buzzard in an important paper on Uniradicular Palsies of the Brachial Plexus.\* In all of these cases there was incomplete paralysis and wasting with diminished electrical excitability of the muscles of the thenar and hypothenar eminences, also of the lumbricals, interossei and long flexors of the fingers, and in all there was an area of relative anaesthesia, "largely dissociative in character," corresponding almost exactly in position with that seen in our case.

The symptoms in these cases and in the case of J. S. are so similar that they must be attributed to a lesion in the same situation. Although different authors are not agreed as to the exact distribution of the first dorsal root yet *both* the anaesthesia and paralysis in these cases correspond in the main very closely to the distribution commonly accorded to it. The absence of any sensory impairment on the hand, and the fact that the motor weakness is incomplete is strong evidence in favour of the view that the lesion is limited to the first dorsal root, *i.e.*, that the first dorsal root is involved before its junction with the eighth

\* *Brain*, Summer, 1902, p. 299.



PLATE 4.



Photographs of the case of lesion of the first dorsal nerve root described in the text (p. 236). The photographs were kindly taken for me by Dr S. A. K. Wilson.





cervical. The absence of oculo pupillary symptoms indicates that the lesion has implicated the first dorsal root beyond the point where the sympathetic fibres leaves it.

The very close resemblance presented by these four cases both as regards the distribution of the symptoms and the degree of paralysis, wasting and sensory impairment seems to afford valuable evidence as to the constancy of a nerve root distribution.

Referring to the etiology of uniradicular palsies of the brachial plexus based upon seven cases which he has personally observed, Buzzard states that "the condition generally occurs in persons who have at some time been the subject of a disease affecting the cardiovascular system. The morbid process is probably vascular in character, and may be sudden or gradual in its onset." Our patient, in whom there was no evidence of cardiovascular disease, attributed his condition to lifting heavy weights (he had frequently in the course of his work to lift a 56 pound weight.)

The pain down the inner side of the forearm from which he had suffered for two or three years before any weakness was noticed in the hand is possibly suggestive of pressure on the root. In this connection it is interesting to note the intimate relation of the first dorsal nerve root to the sharp internal border of the first rib. Such a relation can hardly fail to predispose this root in an especial manner to traumatism.

1st rib  
2nd rib  
3rd rib  
4th rib  
5th rib  
6th rib  
7th rib  
8th rib  
9th rib  
10th rib  
11th rib  
12th rib

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## Abstracts

### ANATOMY.

**INVESTIGATION INTO THE FOREBRAIN OF BIRDS.** L. (135) EDINGER, A. WALLENBERG, und G. HOLMES, *Abhandl. d. Senckenberg. naturforsch. Gesellschaft*, Frankfurt a. M. 1903.

THIS monograph of nearly 100 octavo pages with seven large plates and several figures in the text, on the forebrain of the bird, is the last in the series of Edinger's contributions to the comparative anatomy of the brain.

The difficulties which lay in the way of the completion of the work were due to the diversity of the individual parts in various

species, and more especially to the trouble in the differentiation of the cortex from the striatum, as neither a definite fibre system nor the ventricle separates them in the greater part of their extent. The material available consisted of the brains of over 60 different species, for the study of which various methods were used, viz., the examination of embryological specimens, for morphogenesis, of foetal, young and adult specimens by Weigert's myelin sheath stain, by degeneration experiments (over 80 in number) and Marchi's method for the finer anatomy of the fibre tracts, and by Nissl's and Golgi's methods for cell form and finer relations.

Hitherto this subject has attracted relatively little attention, but the chief results and conclusions of the earlier workers are first briefly abstracted.

In early embryonic specimens the basal ganglia lie free within the ventricle arched over by the pallium, and it is only by the partial occlusion of this through the further growth of the former and the consequent fusion, to a great extent, of the two primary subdivisions of the forebrain that the difficulty in separating them later arises.

The olfactory apparatus is very poorly developed. The anteriorly placed lobes are small, and only a few scattered fibres which soon end in the base of the brain represent the myelinated olfactory tracts, while no definite homologue of the fornix or cornu Ammonis could be found.

The pallium is best developed on the dorsal three-quarters of the hemispheres. It is absent on the base, and generally very atrophic on the median surface of the brain. It is only dorso-medially and over the occipital pole that it is separated from the striatum by the ventricle, and owing to the intimate fusion of these two structures elsewhere, it is very difficult, even by degeneration experiments, to determine whether it is with the one or the other that tracts ending or arising near the surface are connected. It seems probably, however, that but few cortical fibres are added to the striatum tracts as they are present in the reptiles.

The best known bundle of the forebrain is the corticofugal tractus septo-mesencephalicus which consists of axis-cylinders of the cells of the median and dorsomedian pallium. Its fibres converge fan-like on the median surface of the brain, united towards its base into a compact bundle, which after having turned dorsalwards round the other forebrain bundles ends in a lateral thalamic nucleus, in the roof of the midbrain of each side, and to a lesser extent in the region of the oculomotor nuclei. The tractus præcommissuralis which also arises with this bundle from the dorsomedian cortex runs ventrally over the chiasma to the hypothalamus. The tractus cortico-habenularis also springs from

the occipital end of the median cortex, and ends as in reptiles and mammals, in the ganglia habenulæ.

The chief connections of the frontal pallium are the tractus thalamo-frontalis, which degenerates centripetally on destruction of the anterior thalamic nucleus, and the tractus fronto-epistriaticus which also receives fibres from the lobus parolfactorius. The latter runs caudalwards on the base of the brain to end in the epistriatum and that part of the hyperstriatum dorsal to it. Besides these, the frontal cortex is connected with the lateral, and in some species with the occipital surfaces, by association tracts.

The parietal cortex receives fibres only from the thalamus and from the frontal pallium, while the existence of any corticofugal tracts from it could not be discovered. The chief fibre system in connection with the occipital pole is the large tractus occipito-mesencephalicus, which, springing from both pallium and striatum, passes medialwards with the anterior commissure, but, before reaching the middle line, turns abruptly caudalwards to end in the nuclei of the midbrain, and to a lesser extent in the medulla.

The basal ganglia are easily subdivisible into four distinct parts. The largest and most dorsal of these is the hyperstriatum, an elongated dorsally convex mass which extends over almost the whole length of the forebrain, and is easily separable from the others except caudally and ventrally where it fuses with the epistriatum. Its ventral border is separated from the mesostriatum by the lamina medullaris dorsalis, whose fibres spring from it as well as from the cortex, and which is consequently an equivalent of the similarly constituted internal capsule of mammals. This ganglion is connected by centripetal and centrifugal tracts with the anterior, dorsal, and round nuclei of the thalamus, and also doubly by tracts running through the ventral part of the thalamus, with the midbrain.

Ventral to the hyperstriatum, within its concavity, and separated from it only by the lamina medullaris dorsalis, lies another massive crescentic ganglion, the mesostriatum. Its enlarged anterior end projects on the base of the brain as the lobus parolfactorius, while an antero-lateral projection forms the nucleus basalis. These two subdivisions of the mesostriatum vary very much in their degree of development, sometimes the one, sometimes the other being the larger. It is from the former as well as from the neighbouring pallium that the tractus fronto-epistriaticus arises. The body of the ganglion is nowhere directly covered by the pallium, being separated from it laterally by the fissura limbica, and medially by the fissura intraventricularis. The numerous fibres which pierce it from the hyperstriatum and

the cortex give it a striated appearance. These collect on its ventral surface into the brachium cerebri, as the medullated fibres which connect the forebrain with the more caudally lying parts, are collectively named. The mesostriatum itself contributes fibres to the various bundles of the brachium.

The nucleus basalis is of special interest, as it is the only part of the forebrain which has discoverable medullated connections with the medulla oblongata, and the more caudal lying regions. These consist of an afferent partially decussating tract (tractus quinto-frontalis) from the sensory nucleus of the nervus trigeminus, which in the brachium runs further ventral than its other tracts, and an efferent bundle with a similar course which ends in the motor nuclei of the medulla, and sends a few fibres into the lateral column of the spinal cord (tractus fronto-bulbaris et spinalis).

The lobus parolfactorius, so named merely from its position, for no connection with the olfactory apparatus could be made out, contributes fibres to the tractus fronto-epistriaticus, and perhaps to some of the bundles of the brachium.

A collection of large cells, the nucleus entopeduncularis, accompanies the brachium right down into the midbrain.

Laterally between the hyper- and meso-striata lies another ganglion, rich in myelinated fibres—the ectostriatum. The earliest medullated fibres of the forebrain are those which connect this ganglion with a nucleus in the base of the midbrain.

The most lateral and caudal subdivision of the basal ganglia is one homologous in position, and in its fibre-connections with that which Edinger has described in fishes, amphibians and reptiles as the epistriatum. On the one hand it receives the tractus fronto-epistriaticus already referred to, upon the other the anterior commissure partly splits up in, partly arises from it. The greater part of this commissure connects the two epistriata, some fibres join the tractus fronto-epistriaticus, representing a decussating portion of it, and others end in the central grey matter of the thalamus.

The nucleus tæniæ which is situated on the base of the brain lateral to the brachium cerebri sends fibres to the ganglion habenulæ.

Each brachium cerebri is easily divisible into a dorsal and a ventral part, which in the thalamus are separated from another by the nucleus entopeduncularis. In all there are at least ten distinct bundles in each, whose origin, course and terminations cannot be fully described in an abstract.

The comparison of the brains of the sixty species examined gave no little difficulty, that of the dove, goose, and parrot differing in both external and internal configuration not less from one another than do those of the rabbit, dog and ape. The forebrain of the

parrot is easily distinguished by the greater bulk of medullated fibres found within it, and in most respects it seems to be the most highly organised.

The avian brain occupies an anomalous position within the vertebrate series, as it is built on a type peculiar to itself. It has closer resemblances to that of the reptilia than to any other class, but the differences are but emphasised by any attempt at comparison of the individual parts. Its chief characteristic is the enormous relative development of the forebrain ganglia.

GORDON HOLMES.

**ON THE ORIGIN OF THE TRACTUS ISTHMO-STRIATUS (OR (136) BULBO-STRIATUS) OF THE PIGEON.** A. WALLENBERG, *Neurolog. Centralbl.*, 1903, S. 98.

THE so-named tract of the bird's brain was previously described as a centripetal bundle, connecting the mid and forebrain.

In the present paper it is shown that the tract takes origin in the nucleus of the sensory trigeminus. A considerable number of fibres at once decussate to run cerebralwards in the contralateral, but the majority remain in the tract of the same side. Some end in a small nucleus in the midbrain, others in the ectostriatum (see abstract No. 135) and the remainder in the basis of the frontal pole, lateral to the lobus parolfactorius. The bundle is renamed from its connections, tractus quinto-frontalis.

The degeneration experiments from which the above has been described showed in addition the course of the fibres which pass from Deiter's nucleus and the nuclei of the processus cerebelli, through the dorsal longitudinal bundle to the oculomotor nuclei, and to the motor cells of the cranial and spinal nerves.

GORDON HOLMES.

**ARE THE CRANIAL CONTENTS DISPLACED AND THE BRAIN (137) DAMAGED BY FREEZING THE ENTIRE HEAD?** JOHNSON SYMINGTON, *Journ. Anat. and Physiol.*, Jan. 1903.

A CONSIDERABLE number of observations on the topographical relations of the brain, such as those in Macewen's "Atlas of Head Sections," are based upon the results obtained by means of frozen sections of the unopened head. Recently the utility of this method has been called in question by Froriep, who from an examination of published drawings of such sections, as well as from his own experiments, has come to the conclusion that during the freezing process the brain suffers serious injury and disturbance of its parts.

The cranium may be regarded as a box with rigid walls having only one aperture (the foramen magnum) of any appreciable size. The contents of this box are the brain and a variable amount of free fluid situated between the skull and the brain or in the ventricles of the latter organ. Froriep says that 79 per cent. of the brain weight is estimated to consist of water, and the free fluid in the cranial cavity may be taken as from 50 to 100 grammes. Water expands when frozen in the proportion of 1 to 1.09, so that the mass displaced from the cranial cavity during the freezing of an unopened head ought to be about 99 grammes. According to Froriep when a head is put into a freezing mixture the superficial parts of the brain will be first frozen, and as these expand they compress the soft, still unfrozen central parts of the brain, so that the pons, medulla and cerebellum are forced downwards and portions of their nervous substance are driven through the foramen magnum into the spinal canal.

This paper contains a critical examination of Froriep's views and also a description of a specimen in which the entire head had been frozen and cut into a series of slabs in order to determine the surface relations of some of the deeper parts of the brain. These sections were exhibited at a meeting of the Anatomical Society of Great Britain and Ireland last July, in order to give the anatomists present an opportunity of judging whether or not the condition of this brain supported the conclusions of Froriep, and this article is illustrated by a photograph of a median section through the third ventricle, the mid- and hind brains and the adjacent parts of the skull selected from the sections then shown.

In the author's opinion this brain had suffered no appreciable injury or displacement.

AUTHOR'S ABSTRACT.

## PHYSIOLOGY.

### ON THE BIOLOGY AND FUNCTION OF THE CENTRAL NERVE

(138) CELLS. P. KRONTHAL, *Neurol. Centralbl.*, Feb. 15, 1903, p. 149.

IN this lecture, which was delivered in 1900, Kronthal assumes as proved the views of Apáthy and Bethe on the structure of the nerve-cell. If these views are incorrect his whole thesis falls to the ground.

He starts with a consideration of the phenomena which follow stimulation of the grey matter of the cortex; these he regards as due to the excitation of the fibres. He cites in proof of this the laws which regulate and can be deduced from the phenomena of stimulation of the grey matter and of a peripheral nerve respectively.



They are, he says, identical. The nerve-fibres, further, neither begin nor end in the nerve-cells. The latter are merely points of exchange. They do not even comply with the necessary qualities which characterise every organism. One of these qualities is the absorption of food material. The relative loss of weight by various organs in the process of death by starvation is cited as a proof that the nerve-cells do not require nourishment. Luciani found that in death from hunger the fatty tissues lost 93 per cent. of their weight, the muscles 34 to 45 per cent., while the central nervous system lost least of all, only 2 per cent. From this Kronthal concludes that the nerve-cells require little or no nourishment, and hence are not true organisms. The same conclusion he thinks must be drawn from a study of the effects of chloroform and other narcotics on the nervous system. He then takes up the question of cell reproduction as another evidence of organic life. He finds no grounds for believing that nerve-cells multiply by any process of division, and from this point of view also concludes that nerve-cells are not organisms. In his opinion nerve-cells are the result of the coalescence of leucocytes. They have consequently no real nervous function. His idea is that they play a purely passive rôle, that they act merely as insulators. The chief part of the nervous system is the nerve-fibre. All processes which are commonly regarded as psychical are properties of the organism as a whole and not of any individual cells.

As already stated these opinions are based on the correctness of Apáthy and Bethe's views on the structure of the nervous system, and these are still regarded with the strongest doubt by many very reliable observers. Even if they are right, many objections may be urged against Kronthal's statements, and these he does nothing to combat. If these can be met, the usually accepted ideas of nervous and mental processes must unquestionably undergo a wholesale revision. JAS. MIDDLEMASS.

**OBSERVATIONS ON SOME SPINAL REFLEXES AND THE  
(139) INTER-CONNECTION OF SPINAL SEGMENTS. C. S.  
SHERRINGTON and E. E. LASLETT, *Journ. Physiol.*, vol. xxix.,  
1903, p. 58.**

THE researches recorded in this paper were undertaken for the purpose of ascertaining the spinal paths along which "aborally-running reflexes" pass. Instances of reflexes are given which disprove Pflüger's "Fourth Law," which is: "Reflex irradiation in the spinal cord spread upwards or anteriorly, *i.e.* towards the medulla oblongata." The paths along which these reflexes may pass aborally are reviewed, and the authors lean to the view that

the "comma bundle," the "septo-marginal tract," and the "sulco-marginal bundle" of Marie arise largely from intraspinal nerve cells.

In experiments on dogs and cats, the authors adopted a method which they term the method of "successive degeneration." Two translesions of the cord are made, allowing an interval of not less than 260 days to intervene between each operation; after the second lesion the cord is examined by Marchi's method, the degeneration due to the first lesion has disappeared, and the degeneration due to the second can be traced. Records of experiments on fifteen dogs are given. In each animal a hemi-section anterior or posterior, removal of part of a segment, or total transection of the spinal cord was performed. At a time varying from 12 to 568 days afterwards, a second lesion was made in a segment of the cord lying below the site of the first lesion. The animal was killed from 14 to 28 days later, and the cord examined by Marchi's method for degenerated fibres caused by the second injury. Diagrams of the resulting degeneration at different levels are given.

In addition the spinal reflexes were examined before operation, then tested again carefully before the second operation and recorded. The authors divide them into two classes: the "long spinal reflex," in which the muscular reaction takes place in a part supplied by a region of the cord other than that presiding over the part to which the sensory stimulus is applied; and the "short spinal reflex," in which stimulus and movement take place in a part of the body governed by the same region of the cord. A number of both kinds of reflexes are described in the normal animal, and the changes produced in them by various spinal lesions noticed. In some cases the blood pressure and effects on it of stimulation of afferent nerves were recorded immediately before killing the animal.

The authors find in conclusion:—

1. Pflüger's "Fourth Law" does not hold good in the mammalian spinal cord.

2. Afferent channels from skin of shoulder are connected by a lateral tract of fibres with efferent channels to muscles of hind limb on same side.

3. Each spinal segment contains many cells whose axons run to practically all the segments behind it.

4. These association fibres are "long" and "short," and run in lateral, ventral, and dorsal tracts, the lateral being largest, and dorsal smallest.

5. Some of the "short" tracts *may* decussate, most do not. The "long" tracts do not decussate.

6. The spinal path followed in the dog's "scratching" reflex agrees with the course of the lateral long association tract. "Long"



uncrossed spinal reflexes may only require an arc of three neurones.

7. Certain spinal reflexes, *e.g.* "scratching" reflex, once started, tend to maintain themselves by local automatic excitation, and show an "alternating" character explicable as outcome of "reciprocal innervation."

8. There is some evidence that the spinal association tracts descend to spinal mechanisms for skeletal musculature; none that they influence vaso-motor reactions.

9. After cervical transection, stimulation of afferent nerves gives good vaso-motor reflexes if time has been allowed for subsidence of shock.

PERCY T. HERRING.

**REFLEX CENTRE AND REFLEX ARC FOR THE PUPIL.** RUGE,  
(140) *Arch. f. Ophth.*, Bd. liv. F. 3.

At present two diametrically opposite opinions are held regarding the position of the centre for the light-reaction of the pupil. Bernheimer and those who agree with him hold that it is situated in the nuclear area belonging to the third nerve, the small-celled mesial nucleus being the precise situation; while another opinion, championed by Bach, is that the position of the centre is spinal, lying in cervical or upper dorsal region. Both views, incompatible though they seem, are founded upon pathological, clinical, and experimental evidence. Ruge has recently repeated Bach's experiments, which consisted chiefly in the observation of the pupillary condition in monkeys, cats, and rabbits after division of the spinal cord at varying levels. The examination of the pupil had of course to be made immediately upon the decapitation, and if the decapitation was done in the ordinary way pupil contraction always resulted. But as it was impossible in this way to avoid leaving attached to the brain a small part of the cord, Ruge proceeded, in a second series of investigations, as Bach had already done also, to destroy this extreme upper end, which thus probably contained the centre for which he sought, and found that when he did so no pupillary reaction occurred. His cases, however, showed extreme variations in regard to the size of the pupil: for example, in one the pupil remained strongly contracted for a number of minutes and then slowly relaxed; in another it at once assumed a condition of medium size; while in a third, maximum dilatation came on at once. By altering somewhat his method of operating he was able to observe pupil reaction even so long as forty seconds after decapitation, which would of course be an absolute impossibility if the centre were situated in the cord. The next point to be settled was: How far up the cord is it possible to make the

section without destroying the pupil reaction? In the rabbit and the cat he was able to perform the preliminary part of the operation in such a manner as to permit of section just above the calamus (rabbit); respiration ceased instantly but pupil reaction was visible fifty-five seconds later. In another experiment (cat) where the line of section was through the fourth ventricle, a reflex was demonstrable for fifty seconds.

Ruge suggests that the Argyll-Robertson pupil of tabes, etc., may be explained by disease of the cord at the level of the communicating fibres to the sympathetic: if these fibres are injured the ciliary ganglion is, it may be, no longer in a physiological state and the reflex may not take place. It is in this way possible, by means of a unilateral cord lesion, to explain that difficult phenomenon, a unilateral immobile pupil.

W. G. SYM.

**CORTEX CEREBRI AND IRIS.** N. MISLAWSKY, *Journ. Physiol.*, (141) vol. xxix., 1903, p. 15.

PROF. MISLAWSKY, experimenting on cats, found that stimulation of the cortex cerebri produced dilatation of the pupil. This occurred after section of the cervical sympathetic, after extirpation of the sup. cervical ganglion, after section of the spinal cord at the first vertebra, and even after section of the medulla oblongata. The dilatation was small, and not accompanied by any projection of the eyeball or retraction of the nictitating membrane. After section of the third nerve, stimulation of cortex cerebri gave no dilatation.

Prof. Mislawsky concludes that the cortex has an active influence on the centre of dilatation of the pupil, and a depressive influence on the tonic action of the centre in the corpora quadrigemina.

PERCY T. HERRING.

**EXPERIMENTAL CONTRIBUTION TO THE STUDY OF THE (142) PITUITARY BODY.** PIRNONE, *Rif. Med.*, 18th and 25th Feb. 1903.

THE author in this paper gives an account of a series of experiments which he has performed in order to determine the function of the hypophysis in the higher animals. He first refers to the work and opinions of Vassale and Sacchi, of Caselli and others, who assert that the hypophysis fulfils a function of the highest physiological importance, viz., that of maintaining an equilibrium of certain agents in the blood, which, if in excess, stimulate certain tissues to an excessive overgrowth, as in acromegaly and gigantism,

whilst their absence leads to cachexia and arrest of development; and that in the conditions of acromegaly and gigantism and in myxœdema, which are all considered to be the result of some endogenous poison, the overgrowth or hypertrophy of the gland, which is so commonly seen, is a true hypertrophy, the result of excessive functional stimulation, and is, in fact, an attempt on the part of the organism to overtake and to neutralise the toxic substances.

On the other side he mentions Friedmann and Maas, Le Monaco and G. van Rynherk, who hold the opinion that the hypophysis is an involuted organ which has no functional importance whatever.

Pirrone, operating on dogs, investigated the results of complete removal, of partial removal, and of electrical stimulation of the hypophysis, and also of wounding the parts of the brain which lie near the sella turcica, leaving the gland intact.

His method of operation was to resect the zygomatic arch, and having dissected down and made an opening through the base of the skull, to raise the dura mater and so reach the gland.

In the six dogs on which he performed total hypophysectomy he found the following results:—a transitory quickening of the pulse, slowing of respiration, and lowering of temperature, a staggering gait, a constant hanging of the head and much psychic depression. About the fourth day rigidity and weakness of the posterior part of the body, and a curving of the spine appeared. These symptoms gradually increased, the animals passed into a state of coma, and died in from eight to fifteen days after the operation.

Partial removal of the gland produced symptoms similar to those mentioned above, but in a less marked degree; they gradually disappeared, and the dogs regained their normal bodily and psychic condition in about four weeks. On the other hand, electrical stimulation caused a slowing of the pulse and dyspnoea. The same result followed stimulation of the neighbouring parts of the brain, *e.g.* the base of the brain, the frontal lobes and the temporo-sphenoidal lobes.

In the fourth series of dogs, in which he wounded some of the neighbouring parts of the brain, he noticed slight rise of temperature and slight motor and psychic disturbances, which disappeared in the course of a week.

In none of the dogs operated on were there any eye symptoms, and the urine, except in one animal, remained normal.

In discussing these results the author divided the symptoms into two classes—(1) those due to injuries of the various structures and their subsequent reaction, and (2) those which depended on the loss of the pituitary gland. In the first class he included the quickening

of the pulse and the slowing of the respiration, and these, he considered, were due to the lowering of the intracranial pressure which followed the opening of the third ventricle during the removal of the gland and the escape of the cerebro-spinal fluid. The transitory character of these symptoms was explained by the closing of the wound of the ventricle and by the return of the ventricular pressure to the normal. The initial lowering of the temperature he attributed to the shock of the operation.

The symptoms which he considered to be the results of the loss of the gland were the psychic depression, the limitation of movement, the disturbance of nutrition, and the cachexia, which invariably led on to coma and death. These are analogous to the symptoms which follow removal of the thyroid gland.

The changes found in the nervous tissues resembled those seen in the toxic conditions, produced by various organic and inorganic poisons, viz. swelling of the nerve cells, with dissolution of the chromophile elements, and the assumption of a vitreous aspect. Extirpation of the hypophysis, then, causes, in the higher animals, a poisoning of the organism, determined by the absence of the internal secretion of the gland whose function it is to neutralise toxic products accumulated in the blood.

Caselli removed the hypophysis in dogs by operating through the root of the buccal cavity, and in all his cases he noticed glycosuria and other diabetic symptoms, which would explain the fact that the animals did not regain their normal bodily and psychic condition. This glycosuria was probably due to an injury to some part of the base of the brain, possibly of the tuber cinereum, which was caused while opening the base of the skull by this method. In one of the dogs operated on by the author there was a slight albuminuria, but nothing abnormal was found in the urine of the others.

R. G. Rows.

## **PATHOLOGY.**

**ON PURE HYPERTROPHY OF THE BRAIN.** K. TSIMINAKIS,  
(143) *Arb. aus dem neurolog. Institut in Wien*, H. ix., 1902, S. 169.

THE brain described was that of an abnormally large-headed child, born after a normal labour. Nothing wrong was noticed till the third year of life, from which age till death the child periodically suffered from attacks of headache and tenderness of the skull. Later attacks of vomiting occurred and the veins of the forehead became distended, but her intelligence and general psychical state

remained good. When eight years old the patient died from scarlatina.

The brain, all parts of which were uniformly enlarged, weighed, after the ventricles had been emptied, 1920 gr. (average normal weight for this age 1350 gr.). The convexity of each hemisphere was covered by dense and opaque pia mater, thickest over the frontal lobes. The central canal of the spinal cord was patent and distended, but there was no proliferation of ependymal cells. The ependyma of the fourth and lateral ventricles was also normal. Microscopical examination of the thickened pia showed it to consist of dense connective tissue only here and there bound to the cortex by a vessel. Although neither caseous foci nor bacilli were found it was regarded as probably of chronic tubercular origin.

Examination of the cortex revealed no abnormality, the tangential fibres were well preserved and the various layers of cells undisturbed, but the pyramidal cells were as relatively enlarged as the whole brain. There was no evidence of a pathological increase of the neuroglia.

After comparison with similar published cases, the conclusion is come to that there was here the rare condition of *hyperplasia cerebri*; the growth of the brain having been facilitated by an unossified skull due to congenital rickets.

GORDON HOLMES.

**THE FORM OF THE DILATED CEREBRAL VENTRICLES IN  
(144) CHRONIC BRAIN ATROPHY. J. O. WAKELIN BARRATT,  
*Journ. of Anat. and Physiol.*, vol. 37, 1903, p. 150.**

THIS memoir which has been preceded in the same Journal by an article upon the form and form relations of the normal cerebral ventricles, forms part of an investigation which the author has made upon the chronic brain atrophy so commonly seen in lunatics, particularly in cases of general paralysis of the insane and senile dementia. The method of examination employed is described and sketches of the dilated ventricles, drawn to scale, are given. In addition to the purely morphological data which the paper affords, a study of the six cases investigated illustrates the essential dependence in size and form of the cerebral ventricles upon wasting of the white matter of the brain mantle, the basal ganglia not taking any considerable part in the atrophy present.

AUTHOR'S ABSTRACT.

**THE MICROSCOPICAL FINDINGS IN FOUR GASSERIAN GANGLIA REMOVED FOR TRIGEMINAL NEURALGIA.** (145) With a resumé of two previously examined. SIDNEY I. SCHWAB, *Journ. Ment. Nerv. Dis.*, vol. xxx. No. 2, Feb. 1903, p. 88.

THE findings in the four cases may be briefly summarised thus:—

CASE I. Patient aged 50. Five years previously, section and evulsion of infra-orbital and infra-maxillary nerves at the infra-orbital and mental foramina respectively.

Marked chromatolytic and other changes in the small ganglion cells occupying the periphery of the ganglion: perinuclear pigmentation in the larger cells of the central parts of the ganglion. Marchi degeneration of the third peripheral branch.

CASE II. Patient aged 55. Probably no previous peripheral operation.

Similar appearance of smaller cells as in previous case; no perinuclear pigmentation. No Marchi degeneration and no changes in the interstitial tissue or vessels.

CASE III. Patient aged 54. No previous operation. Definite and profound changes throughout the ganglion. Perinuclear pigmentation. Blood-vessels and connective tissue normal.

CASE IV. Patient's age not given. No previous operation. Chromatolysis and vacuolisation seen in every cell; great irregularity of staining. No important pigmentation. No evidence of disease in vessels or interstitial tissue.

The perinuclear as opposed to the ordinary peripheral pigmentation of a cell is, in the author's opinion, probably pathological.

Dr Schwab thinks that there is enough evidence on clinical and anatomical grounds to justify the conclusion that the seat of the lesion in trigeminal neuralgia is in the Gasserian ganglion; that the changes he has found in six ganglia are of sufficient importance to bring them in relation to the symptoms of the disease; and that the cell changes mentioned above should be regarded as the effects of abnormal cell activity rather than as the cause of the neuralgia.

E. FARQUHAR BUZZARD.

**ALTERATION IN THE NERVE CELL IN ACUTE AND CHRONIC (146) IODOFORM POISONING.** R. GIANI ED E. LIGORIO, *Riv. di Patol. nerv. e ment.*, f. 9, 1902, p. 390.

FOR their research the authors chose old animals in preference to young ones on account of the former's greater susceptibility to the poison. In some instances an emulsion of iodoform in glycerine, in others an emulsion in olive oil was used. In acute poisoning three times the toxic dose was injected; in chronic 4 to 5 times

less than the smallest lethal dose was given every 3 to 4 days so as to keep the animal under the influence of the poison. The animals acutely poisoned died in from 30 to 40 hours, while those poisoned more slowly died in from 45 to 60 days. The cerebrum, cerebellum, cord, and spinal ganglia, were examined by Nissl's, Cox's and Weigert's methods.

Five animals were subjected to acute poisoning, and in each the appearances were identical and as follows:—In the spinal ganglia no normal cells could be seen. The pericellular lymph spaces were increased, the chromatolysis was of so advanced a type as to give to the cells a homogeneous appearance and the nucleus in them stained very deeply. In the spinal cord the degree of change was much less, the cells showing a partial irregular chromatolysis with no nuclear change. The white fibres were quite normal. In the cerebellum, Purkinje's cells were pale with much disintegrative change; and occasionally some were homogeneously stained with a shrunken deeply stained nucleus. The large cells of the granular layer showed the same changes, and in addition sometimes vacuolation. In the cerebral cortex the motor cells were all more or less altered. In the first two cases the chromatolysis was peripheral and incomplete, but the smaller cells were more profoundly affected and their nuclei were shrunken and deeply stained. In other two cases the motor cells showed a fine powdery chromatolysis and a homogeneous shrunken nucleus.

Cox's method showed nothing abnormal.

Four animals were injected with iodoform emulsion in doses sufficient to produce chronic poisoning. In the spinal ganglia the pericellular lymph spaces were increased and slight chromatolytic changes were seen in the cells, unaccompanied, however, by any nuclear change. In the spinal cord the changes were slight in some of the cells, but in others a peripheral chromatolysis was evident.

In the cerebellum the cells of Purkinje were normal, but in the cells near them complete chromatolysis could be seen. In many of the giant and large pyramidal cells of the cerebral cortex there was advanced change of the Nissl bodies with destruction of the achromatic network. The small cells were enormously swollen. Many large pyramidal cells showed very little change, the elements being only a little smaller and more confluent.

At the end of their paper the authors point out how some cells show very slight changes after poisoning, and they are of opinion that these have either reacted very little to the intoxication or after having reacted early have become repaired. They draw attention to the numbers of cells spared in the chronic intoxication, and to the grave and diffuse changes in all the central nervous system in acute poisoning.

DAVID ORR.

**PATHOLOGICAL CHANGES IN THE MEDULLA OBLONGATA**  
(147) **IN ACUTE DIPHTHERITIC TOXAEMIA.** CHARLES BOLTON,  
*Arch. of Neurol.*, vol. ii. p. 806.

THIS paper is based on the examination of thirteen cases of diphtheria all of which died during the acute toxic stage of the disease. The first eleven cases died of cardiac failure, and the microscopic appearances in all of them were very much the same. In all these was a well-marked degeneration in the ganglion cells of the nucleus ambiguus. It affected but few of the cells at the lower end of the nucleus, but on passing upwards the degeneration affected a much larger proportion. The other nuclei of the pons and medulla were practically normal. In all these cases the heart muscle showed abundant evidence of fatty degeneration, but the vagi nerves gave no degenerative reaction with Marchi's stain. In the other two remaining cases, which died of asphyxia, the nucleus ambiguus was quite normal and the heart muscle showed no fatty change whatever. The author concludes, therefore, that the changes in the nervous system in cases of diphtheria will vary in different cases, depending on the symptoms and on the stage of the disease. This explains the varying results obtained by other workers on the subject. It is probable also that the cardiac fibres of the vagus originate in the upper part of the nucleus ambiguus and in the nucleus centralis inferior, these being the cells most affected in the eleven cases dying with cardiac failure.

W. K. HUNTER.

**CLINICAL NEUROLOGY.**

**CONTRIBUTION TO THE DIFFERENTIAL DIAGNOSIS OF**  
(148) **NEURITIS (AND NEURALGIA).** H. OPPENHEIM, *Journ. f.*  
*Psych. und Neurol.*, Bd. i., 1902, S. 129.

OPPENHEIM has found that a diagnosis of neuritis is often erroneously made in cases with pains which are really of hysterical or neurasthenic origin, and that this error has had a very unfavourable effect on the course of such cases. In almost all those cases in which neuritis has been wrongly diagnosed, the only objective symptom has been *tenderness of the nerves on pressure*. As the result of the examination of a large number of cases of hysteria, neurasthenia and hysteroneurasthenia (free from alcoholism and any sign of organic disease), he has come to the conclusion that the mechanical excitability of the sensory nerves is frequently



increased in neuropathic individuals, apart from any inflammatory changes in the nerves.

He examined 180 cases from this point of view—confining his attention mainly to the nerves of the arms—and found that 36 of these exhibited undue susceptibility to pressure on the nerves, as indicated either by localised pain at the point of pressure, or, much less often, by paræsthesiæ radiating along the nerve tract. An interesting point brought out by this investigation is that the undue excitability of the sensory nerves is not always general in distribution, but may be confined to certain nerves.

Oppenheim recognises that, as there can be no exact measure of the amount of irritation applied or of the pain caused, his results have only a relative value, but he thinks that they prove that in neuropathic individuals the nerves may be unduly susceptible to mechanical irritation, just as the inflamed nerves are in neuritis, and that, therefore, *tenderness of the nerves on pressure cannot be considered as sufficient datum to found a diagnosis of neuritis on.*

He thinks it probable that the increased excitability of the sensory nerves is of central origin, and not due to any abnormal condition of the peripheral nerves themselves. One can readily understand, on this view, that the hyperæsthesia may be increased or localised from various influences acting from without or from within, *e.g.* trauma, infectious diseases, mental influences such as suggestion, etc.

ASHLEY W. MACKINTOSH.

**SUTURE OF THE BRACHIAL PLEXUS IN BIRTH PARALYSIS**  
(149) **OF THE UPPER EXTREMITY.** ROBERT KENNEDY, *Brit. Med. Journ.*, Feb. 7, 1903, p. 298.

AN account is given of three cases of Duchenne's Birth Paralysis of the Upper Extremity, which were treated by exposing the brachial plexus, excising the cicatrix from the affected nerve trunks, and reuniting by means of suture.

The operation is advisable on account of the poor results which have hitherto been obtained in these cases by means of the treatment usually adopted, and is recommended to be undertaken in all cases which, after the lapse of not less than two months, show no sign of spontaneous improvement, such as the development of the faradic responses in the affected muscles. In those cases in which faradic irritability develops in the affected muscles, further delay is recommended in order to give such cases a chance of further spontaneous improvement.

In the three cases described, the muscles affected consisted of the supraspinatus, infraspinatus, teres minor, deltoid, biceps, brachialis anticus, and the supinators longus and brevis, and in

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consequence the patients could not abduct the arm, or flex the forearm, or supinate the hand, and the arm hung by the side in a position of inward rotation with the forearm pronated.

At the operation in each case the lesion was found to be seated at the point of junction of the anterior primary divisions of the fifth and sixth cervical nerves, and consisted of a dense and bulky cicatrix with adhesions to the neighbouring structures. The cicatrix was excised and the two proximal ends united to the three distal ends, viz., suprascapular nerve, branch to the outer cord, and branch to the posterior cord of the plexus.

In the first case the patient was two months old when submitted to operation. He was a first child and the presentation had been cranial and forceps had been used. No improvement had been noticed before operation, and the faradic responses were absent in the affected muscles. Two and three quarter months after the operation the child was observed to be making movements with the arm which had not previously been attempted, and at six months the movements of the arm were fairly well restored. At nine months, when the case was last examined, the movements were almost normal.

In the other two cases, sufficient time had not elapsed from the date of operation for the development of voluntary responses in the muscles, but in both cases improvement was already exhibited in the electrical responses. AUTHOR'S ABSTRACT.

#### **ON TRUE AND APPARENT SERRATUS-MAGNUS PARALYSIS.**

(150) M. BIRO, *D. Zeitschr. f. Nervenheilk.*, Bd. 23, 1903, p. 278.

THE author records two cases of paralysis of the serratus-magnus, the first being of special interest, as it was apparently an example of the rare condition of functional serratus palsy.

The patient was a strongly built, well-nourished man of thirty-one, who complained that his right shoulder-blade projected unduly from his thorax and caused him inconvenience. The condition had been present for eighteen months and was attributed to a strain.

On examination it was found that the inner edge of the right scapula projected markedly from the thorax, especially when the upper arm was flexed in the sagittal plane to an angle of sixty and the forearm to an angle of eighty. The displacement, however, was found to occur only occasionally, even when the arm was put in the above position, and when it did not occur the patient made evident efforts to produce it. Other signs of serratus paralysis were absent; the muscle reacted normally to electrical stimulation, and was not atrophied. It was noted that the scapula was freely

movable when in its abnormal position. Biro regarded the condition as functional because of its inconstancy, because the scapula was only displaced in a certain position of the arm, and because the other signs of paralysis were wanting.

The second case presented the features of a typical case of serratus paralysis, a point of special interest being that it was due to a wound above the left clavicle, which at first caused almost complete loss of sensation and power in the arm. The arm gradually recovered, and ultimately paralysis of the serratus-magnus proved to be the only permanent lesion.

In addition to recording the above cases Biro discusses at some length the mechanism of the scapular displacement in serratus paralysis, and also the various explanations which have been advanced of the now well-known fact, that in many cases the patients can raise the arm on the affected side above the horizontal.

In considering the etiology of the condition the author lays stress on the fact that the position and course of the long thoracic nerve specially expose it to injury. He describes the nerve as usually arising by two roots, from the fifth and sixth cervical nerves, which pass through and unite in the substance of the scalenus medius muscle. No mention is made of the third root from the seventh cervical nerve, which is almost constantly present and which passes down in front of the scalenus medius to join the rest of the nerve generally opposite the first digitation of the serratus-magnus.

The paper is illustrated by three photographs of the second case, and will be found to present a fairly complete survey of recent views on paralysis of the serratus-magnus.

J. W. STRUTHERS.

**THE INNERVATION OF THE TRAPEZIUS.** SCHULZ, *D. Zeitschr.* (151) *f. Nervenheilk.*, Bd. 23, p. 125.

AFTER recording a case of paralysis of the trapezius muscle, Schulz analyses thirty-four other reported cases with a view to ascertaining the exact distribution in the muscle of the spinal accessory nerve and the branches from the cervical plexus.

He divides the cases into three groups. First, those due to extra-cranial injury of the spinal accessory nerve above its junction with the branches from the cervical plexus; second, those due to intra-cranial and medullary lesions; and third, those due to injury of the nerves in the neck, in the region where the spinal accessory is joined by the branches from the cervical plexus. For purposes of description he divides the trapezius into a clavicular, an acromial,

and an adductor portion, and he finds that in the first group of cases, in which the spinal accessory alone was involved, the adductor portion of the muscle was completely paralysed, while the remainder of the muscle was only partially affected, the clavicular portion more than the acromial.

In cases belonging to group three, in which the nerves to the trapezius from the cervical plexus were probably involved along with the spinal accessory, the acromial portion of the muscle was markedly involved. Cases due to intra-cranial and medullary lesions were of no use in deciding the point in question, for in them there was no evidence to show that the lesions might not have affected the nuclei of the cervical nerves as well as of the spinal accessory nucleus. From his examination of the cases Schulz concludes that the adductor portion of the trapezius is entirely supplied by the spinal accessory, the clavicular portion mostly by the spinal accessory and slightly by the cervical plexus, and the acromial portion mostly by the cervical plexus and slightly by the spinal accessory. Schulz's own case was one of partial paralysis of the trapezius attributed to a severe bruising in the left supra-clavicular region which probably affected the spinal accessory nerve at the edge of the trapezius. The paralysis of the adductor portion was complete, of the other portions only partial. The author describes the resulting displacements and discusses the relative influence of paralysis of each of the three portions of the muscle in their production.

J. W. STRUTHERS.

**ON SOME CASES OF TABES IN EARLY LIFE.** OTTO MAAS,  
(152) *Monatsschr. f. Psychiat. u. Neurolog.*, 1902 (aus der Poliklinik  
von Professor H. Oppenheim in Berlin).

In this paper the author describes five cases of tabes. The patients were from 16 to 22 years of age when the first symptoms appeared; all were females. In the first three cases there were definite signs of congenital syphilis; in the remaining two cases, in which there were no indications of congenital specific disease, the patients were married, in both instances syphilis was denied by the patient and her husband, and there were no discoverable signs of the disease. Idelsohn has remarked on the circumstance that the majority of reported cases of juvenile tabes have occurred in females. The cases here recorded bear out this observation, although the total in the literature is as yet too small to warrant definite conclusions. Reference is made to several cases reported during 1902. A complete bibliography is not given, but may be found in a paper by von Halban, which has recently appeared (*Jahrbuch für Psychiatrie*, Bd. xx.).

EDWIN BRAMWELL.

**TABES IN HEREDITARY SYPHILIS (HEREDITARY TABES).**

(153) J. BABINSKI, *Extrait des Bull. et Mem. de la Soc. méd. des Hôp. de Paris*, Oct. 24, 1902.

BABINSKI remarks on the apparent rarity of this condition, and gives references to the twenty-one cases hitherto recorded. He is of opinion that *tabes hérédosyphilitique* is apt to escape recognition unless especially looked for, since the disease in the child often manifests itself as a *forme fruste*.

The author describes two cases.

CASE I. A female, aged 22. Her mother had acquired syphilis shortly before the patient's birth. The patient was an eight months' child. When four weeks old, she had some ulceration about the anus. At the age of 18 she had had interstitial keratitis. The teeth presented the characteristic features described by Hutchinson. The pupils contracted neither to light nor on accommodation. The left knee-jerk was almost absent, the right being normal. The patient suffered from lightning pains. There were no mental alterations. Lumbar puncture demonstrated the presence of a lymphocytosis. The patient's father had preataxic tabes, and details of his case are given; her mother showed no signs either of syphilis or of organic nervous disease.

CASE II. A girl, aged 15½. The patient was born at full time, but did not walk until two years of age. She had had convulsions when 15 months old. Her intelligence had always been somewhat defective, although there was nothing abnormal with regard to her physical development. Six months ago she developed mental symptoms, a period of depression succeeded by intellectual enfeeblement and excitement. There were no hallucinations and there was no delirium. The memory was unaffected, but she had no sequence of ideas. She was loquacious, her conversation was childish, and she laughed incessantly. It was impossible to fix her attention. The teeth were badly shaped, but not of the typical Hutchinson type. The pupils were large and fixed. Ophthalmoscopic examination showed a syphilitic choroiditis. The tendon reflexes of the lower extremities were abolished. She had frequent incontinence of urine. In this case also the cerebro-spinal fluid exhibited a lymphocytosis.

The father of this patient was examined by Babinski, and found to be suffering from tabes; the mother was not seen.

Both these patients were the subjects of congenital syphilis; both had undoubted tabes; and in both instances the patient's father had the same disease. The mental symptoms in the second case pointed to dementia precox or diffuse meningo-encephalitis.

In the discussion which followed, M. Souques referred to a

family consisting of four persons, of whom the father had died of general paralysis, the mother had advanced tabes, and the two daughters both presented symptoms characteristic of the latter disease.

EDWIN BRAMWELL,

**SOME CASES OF HEREDITARY ATAXIA OR FRIEDREICH'S (154) DISEASE** MANNINI, *Rif. Med.*, Feb. 18, 1903.

IN this paper we have the histories of three cases of Friedreich's disease, which occurred in the second, third, and fourth children of a family of ten, of which family four others died in early childhood.

In all three the disease began with a difficulty in walking and an ataxia of the upper limbs. They all stood in the attitude which is typical of this disease, *i.e.* with their feet far apart and turned outwards, and with the head and body bent forwards. In all there was slowness and hesitancy of speech, and the knee-jerks were absent. The later secondary symptoms, such as deformity of the vertebral column and of the feet, paraplegia, and Romberg's symptom, were seen in different degrees in the three cases, according to the length of time the disease had existed.

In all there was almost an entire absence of subjective pain, of any affections of the special senses, of any trophic or sensory disturbances, of loss of muscular sense, and of spontaneous tremors; the eye movements were normal, so also were the superficial reflexes; the plantar reflex was excessive.

True nystagmus was not seen in any case, although in the oldest case nystagmiform jerks were noticed if rapid movements were made.

Mentally the children were fairly intelligent, but shewed evidences of moral imbecility.

In the oldest case the disease began at the age of 13, and at the age of 19 he was unable to stand or use his arms. This inactivity led to a muscular dystrophy and to deformity of the feet. The third child, a girl aged 15, had been affected only a few months, and the signs, although distinct, were not far advanced.

The second case, aged 20, showed the first signs when 17 years old, and he gave a picture of the disease intermediate between the other two. In his case there was slight paræsthesia of the soles of the feet.

As regards the family history, one uncle was an epileptic, the father drank and showed some signs of degeneracy (facial asymmetry and strabismus); there were no other cases of this disease in the family.

The staggering ambulatory gait, resembling that of a man

intoxicated, and the ataxia of the upper limbs, are symptoms which are seen in Friedreich's disease, in the hereditary cerebellar ataxia of Marie, and in cases of gross lesion of the cerebellum. The usual signs of gross lesion of the cerebellum (vomiting, pain, fever, and increased tendon reflexes) were absent; and the increased patellar reflex, the spasmodic movements, the eye disturbances (amblyopia, dischromatopsia, and loss of light reflex), which are seen in the hereditary cerebellar ataxia of Marie, were also absent. In these three cases, however, the nervous elements, which govern co-ordination, were attacked in their course through the spinal cord, and, as a result of this, we find the symptoms characteristic of Friedreich's hereditary ataxia; but in the cerebellar hereditary ataxia of Marie these nervous elements, governing co-ordination, are attacked in the cerebellum.

R. G. Rows.

**CONTRIBUTIONS TO OUR KNOWLEDGE OF HEREDITARY  
(155) DISEASES** (Dritte Mittheilung). Prof. Dr ERNST JENDRASSIK,  
*D. Ztschrift. f. Nervenheilk.*, Bd. 22, 1902, p. 444.

THE paper deals with hereditary disease in its various forms, and the writer considers that the present classification of diseases of hereditary nature is unsatisfactory and gives a narrow view of the whole question.

The hereditary affections are more closely related to one another than they are to certain diseases produced by exogenous causes to which their symptoms bear an apparent similarity, and hence the hereditary diseases should be classed together, though it must be remembered that the variations in the types of hereditary diseases surpass in the multiplicity of their symptoms those which are seen in diseases of acquired origin.

If, for example, the muscular dystrophies are considered, the variety of types recorded are already so numerous and pass so gradually from one into another without any distinct line being able to be drawn between them, that it is impossible to group them into "types."

The author first describes a group of cases in which from hereditary causes there is shortening of the muscular tendons, ligaments, and deformity of the bones.

The first series of this group were three members of a family, an uncle, a nephew, and a niece, who all manifest the same peculiarity, viz., contraction of muscles and loss of knee jerk.

The uncle, a man aged 30, had never been able to walk properly. He had weakness of the shoulder and hip muscles and contraction of the muscles of the arm which commenced when 14 years old. The feet showed a condition of pes cavus. The knee jerks were

absent. The faradic excitability of the muscles was practically unaltered.

The boy, aged 12, learnt to talk at one year old, walked somewhat later and always in a peculiar manner owing to the contraction of the tendo Achillis. The weakness was most marked below the knees. The knee jerks were normal, the feet in position of pes cavus. The condition was slowly progressive.

The girl, aged 9, showed the same weakness as her brother, which commenced when 7 years old and steadily progressed. The arms became affected somewhat later and the elbows became flexed. The knee jerks were feeble and the feet in position of pes cavus.

The second series of this group consisted of four cases, members of one family, with shortening of the muscles of the lower extremity, tremor of the upper extremity, nystagmus and increased knee jerks.

The eldest, a man aged 37 years, was apparently normal both in intellectual and physical development till 12 years old. He then developed tremor of the hands and difficulty in walking. The condition progressed so that there was unsteadiness of the whole body and he became completely bedridden. Whilst in bed there was little movement, but attempts to speak caused the whole body to shake. The tremor affected the large as well as the small muscles. The reflexes were exaggerated.

The second member of the family, a woman aged 25, was first affected when 10 years old. She then began to walk on the tips of her toes. There was tremor of head and arms and marked nystagmus. There was curvature of the spine, and the position of the feet was like to that seen in Friedreich's disease. All the reflexes were active.

The third member of the family, a man aged 21, first showed signs of the disease when 6 years old. There was nystagmus, slight tremor of the hands, stiffness of the jaw, curvature of the spine—the lower extremities were wasted, rigid and adducted. The reflexes were exaggerated.

The youngest member of the family, aged 18, first showed weakness in walking when 6 years old. Horizontal nystagmus was present, the girl stooped and had difficulty in walking. The reflexes were active, ankle clonus and the Babinski response were present.

The third series of this group was characterised by general dystrophy, shortening of tendo Achillis and loss of knee jerk.

A child aged 8 years, was quite normal till 2 years old when she had diphtheria, and following that disease, contraction of the tendons. When 4 years old she had scarlet fever, and after that the disease again progressed. Both feet were in a position of pes



equinus and there was lordosis. The muscles of the upper arm and thigh were wasted. The knee jerks were absent and the electrical excitability of the muscles to faradism diminished.

The fourth series of this group was characterised by general universal dystrophy with curvature of the spine and shortening of certain muscles.

A boy aged 12 years, was the second of a family of 4 children, the others being healthy. The boy had never walked properly and when 6 months old the spine was noticed to be unusually flexible. He did not learn to walk till 3 years old, and the disease progressed so that at 9 years of age he was no longer able to walk. All the muscles were wasted, but there was not complete loss of power in any. The spine when sitting was bent laterally to nearly a right angle. The arms and legs are both in a flexed position and could not be fully extended owing to the contraction of the flexor tendons. Intelligence was good. Sensation was natural. The knee jerks were absent. There was no fibrillary tremor.

The fifth series of this group was characterised by general dystrophy, marked scoliosis and partial muscular contraction. Two members of a family were affected.

A boy aged 16 who had never walked well, but whose lateral curvature was said to have come on rather rapidly when 12 years old. There was general weakness of arms and legs. The knees were contracted and the legs could not be fully extended. The knee jerks were absent.

A girl aged 11 who had only learnt to walk when 3 years old and had always been weak. The shortening of the right leg was noticed when 2 years old. There was marked scoliosis, the muscles were weak and she was unable to rise from the floor, but was able to support her own weight. The tendon reflexes were abolished. The electrical excitability of the muscles was diminished.

The sixth series of the group was characterised by considerable muscular contraction, marked kyphosis, and a pseudo-nystagmus. Two members of a family were affected, a brother and a sister.

The sister, aged 29, was healthy till between 5 and 6, when contraction began in the legs, and when 10 years old she was unable to walk. Nystagmus was present on lateral deviation of the eyes. There was marked curvature of the back and prominence of the sternum, and the muscles of the arms and legs were contracted. The knee jerk was present on the right side, but absent on the left.

The brother, aged 14, was healthy till 8 years old, when the contraction of the legs developed somewhat suddenly after a long walk. There was slight scoliosis of the spine, marked prominence

of the sternum, and contraction of the legs. There was nystagmus on lateral deviation of the eyes.

The seventh series of the group was characterised by deformity of the feet, kyphosis and scoliosis, shortening of muscles, tremor, disturbance of speech, and failure of sight. Two boys in a family of eight were affected.

The elder, aged 18, first had difficulty in walking when 8 years old, and when 10 he was unable to walk and his sight began to fail. There was intention tremor of the hands. The knee jerks were absent, and the Babinski reflex was flexor in type.

In the younger brother, aged 8, the first symptom was failure of sight when 6 years old, followed by weakness of the legs, which progressed rapidly, so that when 10 he was unable to stand. The knee jerks were exaggerated. The ocular movements were good except for a slight strabismus.

The eighth series of the group was characterised by acquired pes varus with atrophy of the calf muscle.

A man, aged 18, when 12 years old, noticed that the legs were becoming weaker, and there was atrophy of the muscles below the knee and deformity of the feet. Two years later a pressure sore developed on the outer side of the foot which was perfectly painless. The movement of the lower limbs was good. The knee jerks were increased and the electrical reactions were normal.

The above 17 recorded cases occurred in 8 families, and there is little doubt of the hereditary nature of the disease in these cases; on the other hand, in only one instance were the parents blood relations. In the following group of 10 cases, however, the parents were all blood relations.

The influence of consanguinity of the parent is very evident in the following case, in which the patient had, instead of eight, only four great-grandparents.

In this case there was general muscular weakness in the form of myasthenia, but without myasthenic attacks. A boy, aged 8 years, was feeble at birth and could hardly suck, and shortly after birth the parents noticed that the child could not open the eyes and often seemed in danger of suffocation. The child learnt to walk and talk when 3 years old. There was complete ophthalmoplegia externa, the muscles of the face were weak. He could not whistle, chewing and swallowing were difficult, and food and fluid easily got into the larynx. The tongue could only just be extruded from the mouth.

The gait was unsteady and the boy was most easily tired. The knee jerks were brisk and the electrical excitability of the muscles hardly diminished.

Mentally the boy was sound. The case probably belonged to the group "dystrophy," but the affection of the eye muscles was a

most rare complication. A description of two cases of ptosis is then given with ophthalmoplegia externa and loss of patellar reflex occurring in two sisters in later life, two cases of spastic paraplegia and two cases of dystrophy in which the parents were closely related.

Lastly, two cases are described of a condition allied to Friedreich's disease occurring in two sisters, in whom in addition to the ordinary symptoms there was loss of outward and downward movement of the eyes, without nystagmus, and a weakness of the shoulder girdle of a dystrophic type.

This symptom-complex resembles one described by other authors.

The following are the author's conclusions:—

(1) Heredity is a definite cause of disease, and produces forms of disease which cannot arise from other causes.

(2) It is not right to accept heredity as a cause of disease only in those cases in which several members of a family are affected by the same forms of disease, for an apparently sound individual of an affected family may reproduce the disease in their descendants.

(3) The hereditary diseases do not manifest themselves by a perfectly typical, sharply defined set of symptoms, but, on the other hand, the symptoms are most varied and appear in almost endless combinations.

The various "types" may well be preserved for practical use, but they must not be regarded as different diseases.

(4) The hereditary diseases may affect any system of the body, either the nervous, the muscular (dystrophy), the connective tissue (obesity and atrophy), the osseous (achondroplasia), as well as the individual organs.

In many cases it is only the disposition to various affections of exogenous origin which is transmitted, while in others it is a direct aplasia, hyperplasia, atrophy, or degeneration.

(5) The symptoms of hereditary affections may show in the same family greater or lesser variation, still the general features of the disease are the same.

(6) Peculiar and unusual grouping of symptoms, which rarely manifest themselves together, of a chronic long protracted course point with great probability to an hereditary degeneration.

(7) The consanguinity of the parents increases in a great degree the possibility of the occurrence of hereditary degeneration.

FREDERICK E. BATTEN.

**FACIO-SCAPULO-HUMERAL HEMIATROPHY.** A. DEBRAY, *Journ. (156) de Neurol.*, Jan. 20, 1903, p. 63.

THE author describes and discusses a case of "Hémiatrophie Facio-scapulo-humérale" in a man of 25, who, till the onset of this trouble, had enjoyed perfect health.

*His family history* reveals no trace of similar disease.

*The first symptom*, weakness in elevating the left shoulder, came on when the patient was 18 years of age, and since then his condition has become gradually worse, so that at the age of 24 he was compelled to give up his work as a wood-turner.

*Clinically*, there was present a marked atrophy of the muscles of the left face, shoulder and upper arm, and to a lesser extent, in the forearm.

Fibrillary tremor was absent.

There was a deformity of the left scapula which had existed for some time, and the author also calls attention to the large size of the transverse diameter of the head, which measured 7.125 inches—a condition which Pierre Marie and Ouanoff have associated with myopathy.

*On examination*, the special senses and sensory systems were found normal.

The wasted muscles responded briskly to direct mechanical stimulation, and the tendon reflexes on the atrophied side were increased. The plantar reflexes are not reported.

*The Electrical re-actions* were peculiar, as the wasted muscles responded to a lesser Faradic current than the normal muscles, and to a lesser galvanic current with no polar change.

The author mentions a very similar case, described by Risso-limo under the name of "Atrophie myotonique," and classed by him as midway between "Progressive muscular atrophy," and "Thomsens' disease."

The presence of an osseous lesion in both cases might account for the wasting in the surrounding muscles. The reception of abnormal sensations in the spinal cord possibly causing a functional affection in the trophic motor cells in the surrounding segments.

The author concludes that the case is due to either an organic or a functional lesion in the anterior horn cells.

In speaking of the electrical reactions, the author quotes Van Hælst, who says that modifications in the electrical re-actions are due to change in the nervous elements rather than in the muscular, and while he does not necessarily accept that opinion, yet he admits that the study of the causation of muscular atrophy and the absence of electrical changes in cases of pure myopathy, seem to support that view.

The author refers to the uncertainty of the re-action of degeneration as a prognostic sign, and urges that it should rather be used as an aid in arriving at a pathological diagnosis.

In referring to *the treatment*, the author mentions that the patient received no benefit from the administration of large doses of strychnine, but that under massage and faradism, combined with a diet rich in albumen and salts, he has markedly improved.

T. GRAINGER STEWART.

**ARTERIO-SCLEROSIS OF THE SPINAL CORD.** WILLIAM HIRSCH, (157) *Journ. Nerv. Ment. Dis.*, vol. xxx. No. 2, Feb. 1903, p. 74.

THE author states that by reason of their less perfect anastomosis, the anterior spinal arteries are more prone to suffer from degenerative changes than the posterior, and that on account of its more direct supply from the abdominal aorta, the lumbar region of the cord is the most likely seat for these changes to develop. In accordance with this theory, the manifestations of the disease are more common and more marked in the lower than in the upper part of the body, and more usually trophic and motor than sensory in character.

The symptoms of spinal arterio-sclerosis are, in the author's opinion, a gradually progressive weakness of the lower extremities accompanied by tremor, changes in the skin and nails, and diminution or absence of the patellar reflexes. Five cases in which this symptom-complex was present and in which no history of syphilis was obtained are quoted as examples of spinal arterial disease, but they are unsupported by post-mortem evidence. A case of general arterio-sclerosis of the central nervous system with autopsy is also described, and in conclusion Dr Hirsch expresses the opinion that some cases of this kind are in their early stages wrongly placed in the category of neurasthenia.

E. FARQUHAR BUZZARD.

**A CASE OF COLLOID DISEASE OF THE BLOOD-VESSELS OF (158) THE SPINAL CORD.** F. X. DERCUM, *Journ. Nerv. Ment. Dis.*, vol. xxx. No. 2, Feb. 1903, p. 65.

A WOMAN at the age of 39, had an attack of influenza followed by uncontrollable diarrhoea lasting for a year. When the diarrhoea ceased, she began to lose power in her arms and gradually developed stiffness in many of her joints and contracture of some of her fingers. At the end of about two years, her condition was one of atrophic palsy of the upper extremities with spastic paraplegia and

contractures ; in addition, she presented marked anæsthesia over a great part of the left arm and left thorax, slight scoliosis and symptoms of sphincter trouble. After being bedridden for a long time, she died, and a post-mortem examination was made under such difficulties that only the spinal cord was secured.

In the upper part of the cervical enlargement, the central part of the cord was disintegrated and softened ; patches of glassy homogeneous infiltration were scattered through the posterior and lateral columns ; the vessels showed marked hyaloid degeneration of their walls and the anterior horn cells were mostly shrunken and atrophied. Below the cervical cord the crossed pyramidal tracts were the seat of secondary degeneration.

In quoting this as an unique case of its kind, Dr Dercum appears to have overlooked the record of a very similar one which is described in *Brain*, vol. xv. p. 411, by Dr Bulloch.

E. FARQUHAR BUZZARD.

**ON THE ETIOLOGY AND PATHOLOGY OF DISSEMINATED  
(159) SCLEROSIS.** Critical Review. R. T. WILLIAMSON, *Med. Chron.*,  
Jan. 1903.

DISSEMINATED sclerosis is apparently more common in England than in the United States. Amongst 13,864 medical in-patients at the Manchester Royal Infirmary (during a period of 10 years) there were 2294 cases of diseases of the nervous system, of which 61 were disseminated sclerosis, 118 tabes and 6 paralysis agitans. Amongst 2870 medical in-patients at the Ancoats Hospital, Manchester, there were 10 cases of disseminated sclerosis. In the United States the disease is apparently much less frequently met with. Among 6000 cases of nervous diseases, Collins met with only 19 of disseminated sclerosis. (The American statistics are also given in the article by Dr Byrom Bramwell, which appeared in No. 1 of the *Review of Neurology and Psychiatry*, Jan. 1903, p. 12.)

The macroscopic and microscopic pathological changes of disseminated sclerosis are described in detail in the original paper. Points of special interest are the vascular character of the lesions, their irregular dissemination, the absence of secondary degeneration in the majority of cases, and the persistence of axis-cylinders and of nerve cells in most of the patches. A striking feature of many patches is their sharply defined margin—the diseased area ending abruptly, whilst the adjacent tissue is quite normal. In addition to patches of sclerosis, others are often met with in which there is degeneration of nerve fibres, whilst the neuroglia connective tissue has increased very little ; also sometimes recent

patches are found in which the changes are very similar to those of cerebral softening.

A point of much interest is the presence of diseased patches in various stages of development in the same case, some patches presenting firm sclerosis, whilst others are soft and recent. This fact shows that some morbid condition persists, which continues to cause the development of new patches long after the onset of the disease.

The various views as to the pathogenesis are discussed. To the writer several considerations appear worthy of careful attention: (1) The possibility of the patches being caused by the infiltration of the tissues with toxic lymph transuding through the walls of the perivascular lymphatics. (2) The probability of the shape and outline of the sclerosed patches being determined by *physical conditions*, since patches of similar form can be produced when stained fluid is allowed to infiltrate the cord (post-mortem) from various points. (3) The probability of a primary altered blood condition.

AUTHOR'S ABSTRACT.

**INSULAR SCLEROSIS: MOVEMENTS AT REST.** BOUCHAUD,  
(160) *Journ. de Neurol.*, Feb. 5, 1903.

SINCE the days of Charcot, the so-called "intention-tremor" has been considered one of the classical symptoms of disseminated sclerosis. Instances, however, have not been wanting which seem to show that the application of the epithet volitional or intentional to this tremor is too absolute. The author describes a case of a boy aged 16, who was first seen in 1896, presenting the following symptoms:—diplopia, right-sided ptosis, and external strabismus; slight nystagmus; scanning speech, later becoming almost unintelligible; stiffness and rigidity of the lower extremities; exaggerated deep reflexes; staggering gait, followed later by complete inability to stand; and the condition of the toes seen in Friedreich's ataxia. There were no sensory disturbances. There was some degree of volitional tremor, but it was masked by incessant involuntary movements, first of the legs, then of the arms, so aggravated that the patient's clothes were worn through by the movements of the hands on the front of the thighs. During sleep, however, these movements disappeared.

The patient was seen again at intervals of two years, presenting on each occasion a marked amelioration of the symptoms, there being finally no nystagmus, no exaggeration of reflexes apart from the knee-jerks, and no involuntary movements whatever. Speech and writing have much improved.

The author excludes ordinary chorea, rhythmical chorea, paralysis agitans, hysteria, tabes, Friedrich's ataxia, and hereditary cerebellar ataxia; and considers his case as undoubtedly one of disseminated sclerosis, characterised by the persistence of the tremor during repose.

S. A. K. WILSON.

# **ATROPHY OF THE CEREBELLUM AND INSULAR SCLEROSIS.**

(161) ANDRÉ THOMAS, *Rev. Neurol.*, Feb. 15, 1903, p. 121.

UNDER the title of "Atrophie olivo-ponto-cérébelleuse" the writer draws attention to a certain class of cases which he would place by themselves as a new type both clinically and anatomically. The author has already published two cases\* and he now adds a third, referring also to a fourth case by Max Arndt.†

The clinical history of the case is detailed and the morbid anatomy is described exhaustively, and there are ten excellent illustrations. It is impossible to do justice to the carefully described anatomical characteristics of the case in a short abstract. The reader seeking further information will find that a perusal of the original communication will repay his trouble. The chief points in this case, as in the other cases quoted, are as follows:—The disease commences in middle life, has no known antecedent and is slowly progressive. The case reported is that of a woman aged 54 years, and the disease had lasted for five years. It is neither an hereditary, a congenital, nor a familial disease. Clinically it is characterised by the slow onset of cerebellar symptoms, intention tremor and nystagmus. A peculiarity of speech resembling that met with in disseminate sclerosis occurs. In the late stages bulbar symptoms, increase of the deep reflexes and bilateral spasticity occur. The clinical aspect is not unlike that of disseminate sclerosis. Pathologically the disease is a primary cell atrophy of the cortex of the cerebellum, the inferior olives and of the nuclei pontis. The atrophy affects the different regions of the cerebellum unequally. There is complete degeneration of the middle peduncles of the cerebellum and partial degeneration of the restiform bodies. Helweg's triangular path descending from the inferior olives is also degenerated.

The superior peduncles and the dentate nucleus and the other central nuclei of the cerebellum are always intact.

JAMES COLLIER.

\* J. Dejerine and Thomas, *N.I. de la Salpêtrière*, 1900. A. Thomas, *Le Cervelet*, 1897.

† Max Arndt, *Arch. für Psychiatrie*, 1894.



**CASE OF SPINAL CORD TUMOUR.** Prof. H. OPPENHEIM, *Berliner*  
(162) *klin. Wchenschr.*, 1902, No. 2.

THE author describes the clinical condition of a man 40 years of age who, when first seen in September 1900, complained only of persistent and increasingly severe shooting pain on the left side, corresponding to the distribution of the eighth and ninth left dorsal roots, and which had persisted for two and a half years. Examination revealed no deformity of the vertebral column, but there was some pain on pressure and on percussion over the fourth and fifth dorsal vertebræ. There was also some weakness of the left abdominal wall, which did not react so well to faradism as the muscles of the right side. The left abdominal reflex was absent. No signs of compression of the cord were present, but a tumour within the vertebral canal was suggested as the probable cause of the condition. As, however, bone disease was a possibility, the patient was ordered a stiff jacket, and operation not then suggested.

Three weeks later the weakness of the left abdominal wall was more pronounced, the left abdominal reflex was still absent, and there was also found dulling of sensibility to touches and pricks in the left hypochondriac region (corresponding to D 8 and 9); some diminution of temperature sense was found on the right thigh. No loss of power could be made out in the lower extremities, but the patient complained of easily becoming tired on walking.

Four weeks later, as the pain and other symptoms remained unaltered, he was put on extensions in the supine position, and from this time forth signs of cord compression rapidly supervened, so that by November 12th (ten weeks after first being seen) the left leg was completely powerless and the right leg was very weak. On the other hand, analgesia was most marked in the right leg.

Operation was now advised and performed by Sonnenburg, who removed an intra-dural myxo-lipoma of about the size of a hazelnut, which was compressing the cord and neighbouring roots on the left side at the eighth and ninth dorsal segments. The symptoms began to improve within twenty-four hours, but the patient died of meningitis on the eighteenth day.

Post-mortem there was found a rather diffuse degeneration of fibres of the cord, especially numerous in the posterior columns, and with the usual secondary degenerations. The author is of opinion that complete recovery might have occurred but for the occurrence of the cerebro-spinal meningitis.

STANLEY BARNES.

**CASE OF SPINAL CORD TUMOUR TREATED BY OPERATION.**

(163) H. OPPENHEIM, *Berliner klin. Wchenschr.*, 1902, No. 39.

THE author describes a girl of 18 who came to him on January 28th, 1902, complaining of pain in the right side, and other symptoms. There was a history of tuberculosis in the family, but none in the patient. For a long time she had been treated for scoliosis, and in August 1901 she began to complain of pain in the right abdominal region. In October 1901 weakness and stiffness of both legs came on, especially in the right. Some transient difficulty with micturition and defæcation were present before she was first seen by the author. On examination, the vertebral column showed scoliosis (to the left), but there was no pain on pressure or on movement. There was a spastic paraplegia, the right leg being the weaker; on the latter side, foot clonus and Babinski's sign were present. The right abdominal reflex was present.

The patient gradually got worse despite medicinal treatment and extension on a sloping bed; by April 15th, 1902, there was found a very severe paraplegia, the right leg being very rigid and almost powerless, whilst the left was also stiff and weak. There was absolute anæsthesia from the level of the umbilicus to Poupart's ligament on both sides, the anæsthesia reaching about 2 cm. higher on the right than on the left. No abdominal reflex was present on either side. Over the legs sensation was almost completely lost, but there was hyperalgesia of the right sole, and cold was on both sides better appreciated than warmth. There was complete loss of sensation of movement at joints (knees, ankles, toes). There was no pain on movements of the vertebral column.

A diagnosis of extra-medullary spinal tumour was made, and laminectomy was performed by Sonnenburg. The 8th and 9th laminæ were removed, and the theca incised, and a fibroma 3 cm. long was easily removed. No complications ensued, and the patient got completely well in about two months.

The author discusses the diagnosis of the character of the disease and of the level of the compression. With regard to the former, he says that the main points against a diagnosis of extra-medullary growth were the absence of severe pain and the presence of scoliosis; but that the regular and rather rapid sequence of root signs, then cord compression signs of the Brown-Séquard type followed by almost complete paraplegia were sufficient to justify a diagnosis of extra-medullary growth.

STANLEY BARNES.

**DISCUSSION ON SPINAL CORD TUMOURS.** *Journ. Nerv. and*  
(164) *Ment. Dis.*, Feb. 1903, p. 100.

At a meeting of the New York Neurological Society on November 4th, 1902, a discussion took place on this subject. Dr Joseph Collins opened the discussion; an abstract of his paper has already appeared in this Review (p. 108).

Dr Joseph Frenkel described a case of fibrosarcoma of the cauda equina. The tumour was removed and the patient improved after the operation, but died about two months later.

Dr Allen Starr referred to a paper, read before the Society in 1895, in which he had analysed the autopsy records of 123 cases of spinal cord tumour. Out of 100 of these in 75 per cent. surgical interference should have proved successful. He drew attention to a recently published paper by Schlesinger, containing records of 400 cases of which in the opinion of that writer 60 per cent. were operable. Of Schlesinger's cases one-fourth were sarcomata. Dr Allen Starr had personally met with ten cases of spinal cord tumour, of which only six had been operated on, and all had died, two of meningitis, two of bed sores, and two of collapse. He attached great importance to early diagnosis and early operation. As to pain in spinal tumours Dr Starr stated that he did not see just how the differential diagnosis from meningo-myelitis could be made unless this symptom was present.

Dr B. Sachs referred to two of his cases, one operated upon two years the other three years ago. Both were doing well. He insisted that operative interference should be urged just as soon as the diagnosis had been made. A rather sudden development of general myelitic symptoms following upon symptoms which had been unilateral for a considerable time, pointed very strongly to spinal cord tumour.

Dr Walton alluded to a case which had been twice operated on with beneficial results though not cure. If neurologists gave more attention to the possibility of the presence of spinal cord tumours the diagnosis in his opinion would be more often made at an early stage.

Dr Robert Abbe mentioned a case which had been operated upon some time previously; the patient was steadily progressing. He remarked that sometimes a tumour of the spinal cord might grow for a long time and yet give rise quite suddenly to symptoms.

EDWIN BRAMWELL.

**EXPERIENCES OF AN EPIDEMIC OF CEREBRO-SPINAL (165) MENINGITIS.** R. TRAVERS SMITH, *Practitioner*, March 1903, p. 338.

THE author presents in an interesting paper, his observations, on 36 cases of epidemic cerebro-spinal meningitis, which were treated in the Hardwicke Hospital, Dublin, during the epidemic of 1900-1901. Most of the patients were between the ages of five and twenty years, and females were attacked in the proportion of two females to one male. The mortality was 38%.

In the majority of cases the onset was sudden, with headache, pain in the neck, vomiting, shivering and general prostration. Sore throat was commonly complained of. The temperature varied from 100°-103° F. and was characterised by great irregularity though tertian and enteric types were sometimes met with. Recrudescence was frequent, and a fall in temperature was by no means a trustworthy indication of a favourable issue.

Amongst sensory symptoms, headache, often frontal, was invariable and was generally accompanied by pain and stiffness in the neck. Pain radiating from the spine, sometimes associated with hyperaesthesia or anaesthesia, was characteristic in some cases.

Tonic spasm of varying severity occurred in all cases, and affection of cranial nerves was present in many. Kernig's sign was always found. The most typical reflex sign was the early or late disappearance of the knee-jerks. In two cases Babinski's sign was present.

Delirium was noted in all but the mildest cases and coma of the typhoid or cerebral compression type usually supervened.

Affection of hearing and retinal changes were rare.

Herpes and erythema were common.

A slow, small and compressible pulse was found in all cases, even when there was no cerebral compression.

Marked emaciation was frequently a prominent sign.

Morphia and lumbar puncture were the most successful means of treatment.

The morbid anatomy revealed inflammation of the pia arachnoid, especially at the base of the brain, and on the superior surfaces of the cerebellum, the lateral ventricles were generally distended with a clear or slightly turbid fluid. The spinal meninges showed similar changes.

As regards bacteriology, the diplococcus intracellularis meningitidis was always found.

The paper concludes with an account of two cases of the chronic type, which lasted respectively four and eight months before a fatal issue occurred.

T. GRAINGER STEWART.

**A CONTRIBUTION TO THE STUDY OF ENCEPHALOMYELITIS  
(166) DISSEMINATA. E. BAUCHE, *Neurolog. Centralbl.*, 1903,  
S. 109.**

THE patient, a single woman, developed in her 27th year an attack of dementia præcox. Five and a half years ante-mortem she began to complain of pain and weakness in the left leg, which, as no sign of organic disease could be discovered, was regarded as functional. These symptoms slowly progressed and three years later she finally took to bed. Five months before death tonic spasm affected the muscles of the left thigh, but it disappeared in chloroform narcosis, and the limb was immediately put up in normal position in a plaster bandage. The skin of the buttock and thigh sloughed under this, leaving a deep septic ulcer. Within the next fortnight paralysis of the lower extremities and sphincters, and anæsthesia on the lower half of the body appeared, and three months later, a few days before death, weakness of the right arm.

In the autopsy numerous small and larger foci, generally with sharply defined margins, were found in the basal ganglia, in the white matter of the forebrain, in pons and in cerebellum, as well as in the spinal cord. None were present in the cerebral cortex. Some were tough, others of softer consistency, and they generally lay round a vessel. In the cord they were as a rule wedge-shaped with apex directed inwards.

Microscopically the myeline sheaths and axiscylinders were found in various stages of degeneration, as were also the cells of any grey matter involved in a focus. The neighbouring vessels were congested, and their adventitial sheaths and occasionally the surrounding brain substance infiltrated with small and large round cells, polynuclear leucocytes, small plasma cells and large granular mast-cells. There was also considerable proliferation of neuroglia, many of whose cells seemed to act as scavengers. The later changes were softening and neuroglial sclerosis with secondary vascular change.

As no sign of organic disease could be found during the five years she complained of pain and weakness in the left lower limb, these symptoms are regarded as functional, and the encephalomyelitis the result of infection of a subnormally resistant nervous system from the extensive septic ulcer.

GORDON HOLMES.

**A CONTRIBUTION TO TUBERCULAR DISEASE OF THE PONS.**

(167) FR. SCHÖELER, *Klin. Monatsblätter f. Augenheilk.*, xl. Jahrgang, Bd. 11, Nov. 1902, p. 313.

THE author describes briefly two cases of tubercular tumour of the pons. He has collected from the literature 140 cases of pontine disease in all of which there was a post-mortem. From an analysis of these cases he concludes that the most characteristic symptom of a lesion of the pons is associated paralysis of the lateral movement of the eyes. In a large number of cases paralysis of the sixth nerve on the side of the lesion was associated with paralysis of the opposite internal rectus, the movement of the internal rectus on convergence being quite good. The paralysis of the internal rectus for lateral movement was not so great as that of the external rectus on the side of the lesion.

Conjugate deviation of the eyes is not uncommon in pontine lesions, and is attributed by v. Monakow to a secondary contracture of the antagonistic muscles. That this is not always the explanation is shown by cases recorded by Marchi and by Hallopeau and Giraudeau, in which the deviation was to the side of the lesion. The conjugate deviation which results from cerebral lesions is a temporary symptom lasting from a few days to a few weeks; while the deviation due to a pontine lesion is persistent. Conjugate deviation of the eyes unaccompanied by other symptoms occurs, so far as the author can ascertain, only in disease of the pons.

The relative frequency of different symptoms in the 140 cases of pontine lesion, which included tumour, hæmorrhage, softening, etc., is referred to in considerable detail.

EDWIN BRAMWELL.

**CONTRIBUTION TO THE SYMPTOMATOLOGY OF PARALYSIS.**

(168) AGITANS. H. OPPENHEIM, *Journ. f. Psychol. und Neurol.*, Bd. i., 1902, S. 134.

OPPENHEIM has had his attention directed recently to *salivation* as a symptom of paralysis agitans, and he believes that it occurs not infrequently, as a rule at the very beginning of the trouble and sometimes even before the more usually recognised symptoms. He gives notes of six cases, in which salivation was one of the most prominent and troublesome symptoms and in one of which (Case II.) it was present for years before tremor appeared.

As regards the cause of the salivation, he thinks that it is mainly a true primary hypersecretion of the saliva, analogous to

hyperidrosis, with which it appears usually to be associated in paralysis agitans. Other possible explanations are that it is due to tremor of muscles of the lips, tongue and jaws, or to rigidity of the muscles of the region of the tongue, palate and throat, with consequent defect in swallowing of the saliva. Such mechanical causes may play a part in certain cases, but in some of Oppenheim's cases there was no tremor of these muscles and in others the salivation appeared before the tremor or rigidity of muscles.

Short reference is made to several cases of paralysis agitans under Bruns' observation, in which marked bulbar symptoms were present in addition to salivation, viz. dysphagia, dysarthria, and disturbance of the movement of the tongue.

ASHLEY W. MACKINTOSH.

**ATTACKS OF GIDDINESS AND UNCONSCIOUSNESS DURING  
(169) LAUGHTER.** H. OPPENHEIM, *Monatsschr. f. Psychiat. u. Neurol.*, Bd. 11, H. 4, 1902.

THE writer records two cases of a hitherto undescribed character, for which he suggests the name of Gelosyncope, or Geloplegia, or Ictus Ridentis; although, until more cases present themselves, he admits his communication to be merely provisional.

One case is that of a healthy, though shy and rather lazy, country girl, aged 18, who for about a year previously had suffered from attacks of sudden unconsciousness during hearty laughter.

The other case is of a Member of Parliament, a strong man, aged 45, non-syphilitic and non-alcoholic, though a heavy smoker. He had had one attack of sudden loss of consciousness while laughing at a joke, and twelve years later two similar attacks.

The cases have these points in common. During hearty, but quite natural laughter as a result of merriment, a sudden and absolute loss of consciousness occurs. The patients become purple in the face, the expression fixed, and the body falls forwards or to the ground. The attack is over in a few seconds, there are no twitchings, no biting of the tongue, no incontinence of urine, and the mental condition is quite natural so soon as consciousness is regained.

The writer suggests that the condition is directly due to the central, psychical condition of excitement which normally discharges itself by laughter.

He differentiates the condition from epilepsy because all the usual symptoms of the latter are absent, while the laughter is always quite natural and therefore not to be regarded as an aura. A few cases of epilepsy induced by laughter, due to tickling the soles of the feet, are recorded by van Swieten, Esquirol, and

Reynolds. Only if one extend the term epilepsy to include every transitory loss of consciousness, can one apply it to these attacks.

Hysteria with its prolonged laughter rising to a spasm or ending in convulsions is totally different, and neither of the cases in question shows any other hysterical symptom.

Organic brain disease is considered but dismissed for want of other signs, and because the attacks are associated purely with laughter.

The swooning of infants which Henoch attributes to excessive crying combined with excitement, and which consists of a spasm of the inspiratory muscles, is admitted to have traits in common with but to be separated "toto cœlo" from the present cases.

A comparison is made with a case of disseminated sclerosis recorded by Oppenheim. This patient took fits of uncontrollable laughter, in which he became cyanotic, breathless, blue over the whole body, and finally completely exhausted.

A close relationship is supposed by the writer to exist between his cases and the laryngeal vertigo of Charcot, which in nervous persons, tabetic cases, and some cases of laryngeal disease produces sudden loss of consciousness, preceded only by a tickling or choking feeling in the throat, and a few coughs, and followed by complete recovery in a few seconds.

JOHN D. COMRIE.

**A NOTE ON GASTRIC TETANY.** B. G. A. MOYNIHAN, *Practitioner*, (170) March 1903, p. 354.

THE author refers to a paper by Dr Carnegie Dickson which appeared in the *Practitioner* of January 1902 on "A case of Gastric Dilatation and Tetany" in which the general view taken was that gastric tetany was a very serious and rare disease.

Mr Moynihan has had personal experience of five cases of this disease, all occurring in males and in which recovery took place after gastro-enterostomy had been performed. He describes the most severe of these cases in which he operated with complete success. In conclusion he urges, that the condition is neither so serious nor so rare as it is supposed to be and claims that the timely performance of gastro-enterostomy would remove the cause of the attacks.

T. GRAINGER STEWART.

**REMARKS UPON TIC.** H. OPPENHEIM, *Journ. f. Psychol. und (171) Neurol.*, Bd. i., 1902, S. 139.

OPPENHEIM emphasises the distinction between simple muscular spasm and tic: the former is a motor act, caused by irritation at any part of a reflex arc, while tic is always a psychomotor act—



either a morbid expression of emotion or a reflex movement which has become a habit and persists after removal of the cause.

Reference is made to the frequency of *enuresis diurna* as a symptom of tic général: Oppenheim has seen several cases in which enuresis was one of the most obstinate and painful symptoms.

As regards *prognosis*, Oppenheim—somewhat in opposition to the school of Charcot—believes that tic is curable, not only in the mild, localised forms, but also in the severe, generalised forms. Notes are given of several cases of the latter forms: in two, recovery was complete, and in a third there remained only an occasional movement of the facial muscles and fingers.

In the treatment of tic, muscular exercises are of great importance, *traitement rééducateur*, and Oppenheim quotes at length from a former work his views regarding the principles of application of such exercises in nervous diseases. The cases in which he uses this method of treatment are those in which there are signs of defective control, a tendency to muscular unrest or to involuntary movements, especially under emotional influences. He has found this treatment successful in many cases of hysteria, neurasthenia, tic convulsif, tic général, localised muscular spasms, etc. One must be careful not to make too great a demand on the attention and energy of the patient.

ASHLEY W. MACKINTOSH.

**ON THE RÔLE OF THE CEREBELLUM IN VOLITIONAL ACTS  
(172) WHICH REQUIRE A RAPID SUCCESSION OF MOVE-  
MENTS (DIADOCOCINESIA). J. BABINSKI, *Rev. Neurol.*,  
Nov. 15, 1902, p. 1013.**

THE author points out that in conscious equilibration there are embodied two distinct entities—static equilibrium, evidenced when the body or one of the limbs is held still, and cinetic equilibrium, which is exercised during the performance of a movement.

He draws attention to the apparent paradox that in subjects where a lesion of the cerebellum or of its peduncles exists, there is a marked increase of static equilibrium co-existing with a marked decrease of cinetic equilibrium in the area of the body corresponding with that part of the cerebellum which is affected. He demonstrates the increase of the static equilibrium in the following way:—

A patient suffering from unilateral cerebellar lesion, lying upon his back, is told to raise one of his lower extremities and hold it still, when the following marked peculiarities are noticeable:—

- (a) There is a remarkable fixity about the limb, corresponding with the cerebellar lesion, as compared with the limb of the

sound side, or as compared with the limbs of a normal person placed in a similar attitude.

- (b) There is absence of oscillation and jerky movement.
- (c) There is absence of that muscular vibration which is usual in a normal limb held rigidly.

In other words, when the attempt to hold the limb rigidly is made, a more complete rigidity is maintained than in a normal limb.

The loss of cinetic equilibrium (diadococinesia) in cerebellar cases is best demonstrated when the rapid performance of voluntary alternate movements is observed. As a typical example, the movements of pronation and supination of the forearm are taken. The patient is told to pronate and supinate rapidly and without interruption. It will be observed that the act is performed at least three times as slowly upon the side corresponding with the cerebellar lesion as upon the opposite side, or as in a normal subject, whereas, on the other hand, a single movement of pronation and of supination is performed as rapidly as in health.

The author thinks that this phenomenon is due to interference with the association of excito-motor and inhibitory impulses, and that the seat of such association is in the cerebellum.

This diadococinesia explains many of the difficulties of movement that the cerebellar patient exhibits, especially those in writing and in walking, and the importance of the true succession of alternate movements in the performance of these acts is obvious.

These explanations of M. Babinski are very suggestive, and it seems very probable that they are in part, at least, correct. They should add considerably to the distinctive signs of cerebellar lesions at present available.

JAMES COLLIER.

**CONCERNING CEREBRAL AND SPINAL REFLEXES.** A. V. (173) KORNILOW (Moskow), *D. Ztschrift. f. Nervenheilk.*, Bd. 23, H. 3 u. 4, 1903, p. 217.

THIS paper deals with certain questions which relate to the reflexes, and which have come into special prominence since the discussion on the subject at the International Congress held in Paris two years ago.

1. Recently described reflexes are first considered:—

Regarding the *scapulo-humeral reflex*, the author's observations are in accord with those of Steinhausen, who came to the conclusion (*Neurol. Centralbl.*, 1901), after examining 300 soldiers, that the scapulo-humeral is a periosteal reflex, that it is best obtained from the internal border of the scapula close to the point where the spine joins it, and that on percussing here a contraction occurs in the posterior fibres of the deltoid. Steinhausen further

states that this contraction is constant in health, although it was unequal on the two sides in 12 per cent. of those persons examined by him. With this latter observation Dr Kornilow does not agree; he is of opinion that perfectly healthy subjects are frequently met with in whom the reflex is not present.

Referring to the *supra-orbital reflex* of MacCarthy, the author discusses the explanations advanced by v. Bechterew and Hudo-vernig to account for the phenomenon. He himself believes that it is neither a skin nor a periosteal reflex, but that it belongs to a special class, to the so-called defensive reflexes (*abwehrreflexen*).

In 1901, v. Bechterew drew attention to an *ilio-hypogastric reflex*. When the inner side of the thigh in the neighbourhood of the inguinal fold is stroked with the handle of a percussiom hammer, a distinct sinking in of the abdominal wall in the supra-inguinal region may be seen. Van Gehuchten, who referred to this reflex in 1900, believed that it only occurred in women and that it was analogous to the cremasteric reflex; Crocq (1901), however, showed that it could also be obtained in men, an observation which the author has confirmed. The hypogastric reflex may have a certain significance in estimating the level of a spinal cord lesion.

2. The *Babinski phenomenon* still excites much interest. Its clinical significance, Dr Kornilow believes, is not yet definitely established; he holds that the term Babinski phenomenon should be confined to "the extension of the great toe alone or in association with extension or flexion of the toes." The author gives his reasons for believing that the phenomenon cannot be regarded as a modification of the plantar reflex. Dr Kornilow refers to four cases of cerebro-spinal meningitis which he has recently examined with Dr Schamschin, in which although the Babinski sign was present during life, no sign of disease was found anywhere in the pyramidal tracts, from the cortex down to the sacral region of the cord. "These cases definitely prove that the Babinski phenomenon may be present where there is not the slightest sign of organic disease in the whole pyramidal tract." We quite fail to follow his argument when he says that because in these cases there was no single symptom present in favour of disease of the pyramidal tract (no paralysis, cramps or contractures and in some cases both the skin and tendon reflexes were absent), therefore the Babinski phenomenon stands in no direct relation to a functional disturbance of the pyramidal tract.

From his own observations, Dr Kornilow admits that the Babinski sign is not met with in healthy individuals, and that it often occurs in association with disease of the pyramidal tracts. He is of opinion, however, that the differential importance of the Babinski sign in the diagnosis of hysteria is not pathognomonic.

The following brief paragraph contains his reasons for this conclusion:—"I have observed two well-marked cases of Hysteria in which during and after the hysterical attacks, in the form of cortical epilepsy (hysterischen Anfällen in Gestalt von corticaler Epilepsie), the phenomenon was distinct. In one of these cases, a boy of fourteen, in consequence of a mental shock, was seized with hemiplegic hysterical attacks of a cortical character (hysterischen Anfällen corticalen Charakters in hemiplegischer Form). I diagnosed hysteria, and this diagnosis was later confirmed. The boy was soon quite well; the Babinski phenomenon was well marked in his case. The phenomenon as a differential sign between hysteria and organic paralysis has no pathognomonic importance. Roth, Cohn and Tempowski endorse this statement."

If Dr Kornilow wishes to convince neurologists that "the phenomenon as a differential sign between hysteria and organic paralysis has no pathognomonic importance," it is hoped that he will record his cases in greater detail in a later communication. These meagre facts completely fail, in our opinion, to carry conviction.

3. The *condition of the reflexes in transverse lesions of the spinal cord in the cervical and upper dorsal regions* is next considered. The observations of Bastian and Bruns on the disappearance of the reflexes in cases of complete transverse lesion are referred to. A number of cases in which, although the cord was completely divided the reflexes were present, are cited as disproving the view that the loss of the reflexes in complete transverse lesions is dependent upon the cutting off of cerebellar-spinal impulses. Bruns has shown that in some cases in which the knee-jerks are absent, the anatomical reflex arc shows no sign of disease. Dr Kornilow mentions a similar personal observation. The paralyses resulting from experimental division of posterior roots are referred to as showing that disturbance of function may arise where no visible defect is to be detected in the nerve paths. Anatomical and physiological integrity do not necessarily correspond. The solution of this question, the loss of the reflexes in cases of transverse lesion, remains for the future to decide.

4. The *nature of the tendon jerks* is discussed at considerable length. The author considers the points which have been raised for and against the view that they are true reflexes. He attaches great importance to the work of Sternberg, with whose opinions he entirely agrees. He concludes that "there is both a bone and a muscle reflex. The reflex nature of the bone reflex is doubtful, that of the muscle reflex probable."

The paper contains many points which it has been impossible to touch upon in this abstract. A considerable bibliography is appended.

EDWIN BRAMWELL.

**ON THE ACROMIAL REFLEX.** VON BECHTEREW, *Neurol. Centralbl.*, (174) March 1, 1903, p. 194.

**ON THE CARPO-METACARPAL REFLEX.** VON BECHTEREW, *ibid.*, (175) p. 195.

IN cases where the reflexes of the upper limbs are exaggerated, as in organic hemiplegia, amyotrophic lateral sclerosis, etc., v. Bechterew describes an acromial and a carpo-metacarpal reflex.

What he terms the acromial reflex is elicited by percussing the acromion and coracoid processes of the scapula, the resultant movement being a slight flexion of the elbow, together with a little inward rotation at the wrist and flexion of the fingers. This last part of the phenomenon only occurs in cases of great exaggeration of reflexes. The muscles chiefly involved in this reflex are the coraco-brachialis and biceps (of which the former arises wholly and the latter partly from the coracoid process). It is recommended that the acromion process be percussed through a pleximeter, repeated direct tapping of the bone being painful.

The carpo-metacarpal reflex is elicited as follows:—The observer lays the patient's hand upon his own, dorsum upwards, with the second and third phalanges of the fingers hanging loosely down. Then with a percussion hammer the carpus and neighbouring part of the metacarpus are tapped, especially towards one or other lateral margin of the limb. The result is a flexion movement of the interphalangeal joints, with the exception of the thumb. In cases with excessive exaggeration of reflexes, flexion of the fingers may also be elicited on mechanical stimulation of other parts of the hand, and even from the flexor tendons. V. Bechterew states that the carpo-metacarpal reflex is the one which is most constantly present in lesions above the cervical enlargement, and that its level corresponds to the first dorsal and lower cervical segments of the cord. It is therefore of some importance in the diagnosis between functional and organic palsies.

PURVES STEWART.

**ON INCONTINENCE OF THE BLADDER AND PARALYTIC (176) PHENOMENA IN THE EXTREMITIES IN FOCAL LESIONS OF THE BASAL GANGLIA.** A. HOMBURGER, *Neurol. Centralbl.*, March 1, 1903, p. 199.

HOMBURGER has made observations upon cases of bilateral foci of softening in the optic thalami and corpora striata. Corroborating Czylharz and Marburg,\* he supports Nothnagel's view that there are sub-cortical centres for the innervation of the bladder located

\* *Jahrbücher f. Psychiatrie*, 1901.

in those ganglia. Unilateral foci of softening produce only transient incontinence of urine for a few weeks, followed by permanent precipitancy and occasional nocturnal incontinence. But if the lesions in the ganglia be bilateral, the result is a permanent incontinence, exactly resembling that due to a spinal lesion—i.e. the bladder automatically, at more or less regular intervals, discharges a fairly constant quantity of urine, yet never completely empties itself, there being always a certain amount of residual urine.

We may therefore conclude that the subcortical innervation of the bladder is a bilateral one. Superficial lesions of the ganglia (lesions situated close beneath the surface of the thalamus or corpus striatum) do not produce incontinence. But bilateral lesions in the substance of the ganglia produce a characteristic clinical picture. The gait is unsteady and stiff, the upper extremities comparatively unaffected, the tendon and periosteal reflexes lively, but ankle-clonus, as a rule, is absent or indistinct. The intellectual functions progressively decay, and there is generally uncontrollable laughing or crying; athetosis is rare. Such cases are distinguished from lesions directly interrupting the cortico-spinal motor path by the absence both of Babinski's reflex and of the tibialis phenomenon.

PURVES STEWART.

**THE REACTION OF NEURO-MUSCULAR DEBILITY.** Examination (177) into the General Conditions of Patients. M. KLIPPEL, *Arch. Gen. de Med.*, Feb. 10, 1903.

In this paper the author discusses the significance of a neuro-muscular syndrome which he finds to be present from a comparatively early stage in a great variety of chronic diseases. The symptoms in question comprise:—

(1) General Pathological Myoidema, or mechanical hyper-excitability of the muscles. This is the same condition that is frequently met with locally in the pectoral muscles in cases of phthisis, but in the cases under discussion it is a general condition, unassociated with local lesions.

(2) Exaggeration of the Reflexes, obtained by percussion of the tendons. These two conditions must not be confused. In trying to produce myoidema, an exaggerated reflex is often obtained, but the author maintains that the two are distinct as to their mechanism, and that the one does not absolutely imply the other.

(3) Diminution of the Electrical Reactions. Diminished excitability to both galvanism and faradism is a well-marked feature, especially in the lower limbs. The reaction may even be nearly abolished.

(4) Tachycardia and Tachypnoea. Tachycardia may reach 140 with the patient resting in bed, and the temperature normal. This may be found in cases of cancer of the uterus, and other conditions where interference with the pneumogastric can be excluded. Tachypnoea occurs in the same conditions as tachycardia, particularly in cancer.

Some, or all, of these signs are so frequently found in cases of chronic disease that the author speaks of them as the neuro-muscular syndrome of debility. Their presence indicates a state of organic malnutrition, and in graver cases the hyperexcitability becomes more and more an indication that the neuro-muscular organs are in danger of destruction.

Do the above-mentioned signs serve to indicate the general condition in acute diseases? Not altogether. In some acute diseases abolition of the reflexes may rapidly follow a passing exaggeration. In others the toxins seem rather from the commencement to paralyse than to exaggerate the neuro-muscular reactions, and this without any grave prognosis of the disease. Where the symptomatic identity between acute and chronic diseases does appear is when the fever has abated, during and after convalescence. The signs of the reaction of debility may persist for a long period, even after the patient feels quite well. Some of them, indeed, may persist indefinitely.

W. B. DRUMMOND.

## PSYCHIATRY.

### THE ALKALINITY OF THE BLOOD IN MENTAL DISEASES

(178) ROBERT PUGH, *Journal of Mental Science*, Jan. 1903.

THE author used Wright's method—titration of a small quantity of blood serum taken up in a capillary tube, with an equal quantity of dilute sulphuric acid of known strength. The process is repeated with stronger or weaker solutions of sulphuric acid till the neutral point, as indicated by litmus paper, is obtained.

In twenty control cases Pugh found the average, normal alkalinity was 1.662, i.e. 1000 c.c. of solution containing this amount of  $\text{H}_2\text{SO}_4$  would neutralise 1000 c.c. of serum.

In mania, during the period of excitement and in *dementia paralytica*, very markedly in connection with the convulsive seizures, alkalinity is reduced.

In epilepsy, alkalinity is diminished between the paroxysms, very greatly diminished immediately prior to the onset of the fits and still further reduced after the fit is over. Pugh states that the higher the alkalinity of the blood in epilepsy the less liable is the patient to have a fit, but that it is impossible to elevate and main-

tain the alkalinity within the physiological limits for any appreciable length of time by administration of drugs.

No information is given regarding the drugs employed.

ALEXANDER GOODALL.

**RESEARCHES INTO THE STATE OF THE BLOOD AND THE**

(179) **URINE IN DEMENTIA PRECOX.** MAURICE DIDE and LOUIS

CHÉNAIS. *Ann. Méd.-Psychol.*, Nov.-Dec. 1902.

THE authors comment on the scantiness of the available information on the composition of the urine and the state of the blood-corpuscles in dementia praecox. Their own research they recognise to be as yet very incomplete, especially as regards the blood. Eighteen cases (11 women, 7 men) were examined by them. As regards the blood; white corpuscles alone are dealt with in the present publication. The following varieties are recognised:—Polynuclear, large mononuclear, small mononuclear, eosinophile. The percentage of these is deduced from a large number of observations. The authors say it is difficult to deduce very definite conclusions from the figures arrived at, as the different kinds of white corpuscles appear to vary in different cases for no apparent reason. Thus, the polynuclear were diminished in 5 cases, normal in 9, and increased in 4. But one conclusion is sufficiently clear, namely, that the eosinophiles were increased in number in 12 cases. Whereas normally the proportion is stated to be 1 per cent., the authors find it to be 3 to 4 per cent. in these cases. It is intended later on to publish the result of observations upon the isotonia of the red corpuscles and the toxicity of the blood-serum. As regards the urine, the following points were investigated:—The density, amount of urea, of phosphates, of chlorides; also the presence of albumen, urobilin, indican, and bile-pigments. The density was notably increased in 13 out of 18 cases. Urea was considerably below the normal amount in 16 out of 18. Phosphates were excreted in nearly normal quantity, taking the average of the 18 cases. Chlorides were excreted in excess in 14 out of 18 cases. Albumen was found in only one case, and then its presence was intermittent. Indican and biliary-pigments were never present. Urobilin was present in only one case.

EDWIN GOODALL.

**A CONTRIBUTION TO THE CHEMISTRY OF NERVE DE-**

(180) **GENERATION IN GENERAL PARALYSIS AND OTHER**

**MENTAL DISORDERS.** By ISADOR H. CORIAT, *Am. Journ. Insan.*, Jan. 1903.

DR CORIAT first gives a short review of our present knowledge of the pathological chemistry of the central nervous system, states



that this is practically confined to the fate of the lecithin, and sums up by saying that chemical analyses of degenerated nerve centres show a decrease of lecithin and the phosphorus and an increase of water, proportional to the extent of the degeneration. The lecithin splits up into (1) glycerophosphoric acid, which is excreted in the urine and helps to increase its organic phosphates, (2) stearic acid, which unites with the glycerol radicles to form the neutral fat which gives the well-known micro-chemical Marchi reaction with osmic acid; and (3) cholin, which passes into the cerebro-spinal fluid and blood.

Dr Coriat considers that the cerebro-spinal fluid, in its rôle of the lymph of the central nervous system, affords a reliable index of the morbid chemistry of the nerve centres themselves, and accordingly he proceeds to give the results of his examination of this fluid in 34 cases, including 14 general paralytics, 6 alcoholics, and other types of mental disease.

The cerebro-spinal fluid was obtained by lumbar puncture soon after death in all cases. Its *amount* varied from 10 c.c. to 150 c.c., being largest in the general paralytics. It was either *colourless or straw-coloured*, unless contaminated with blood. It was *clear or opalescent*. The *specific gravity* in 6 cases varied from 1006 to 1010. The *reaction*, in 8 cases, was neutral in 2 and slightly acid in 6, the *acidity* being due to lactic acid (optically inactive) which the author regards as a product of prolonged activity of the nerve centres. A *reducing body*, tested for in 5 cases, was present in 4. *Cholin* was present in 23 out of the 34 cases, namely in 14 general paralytics, 4 alcoholics, 2 cases of dementia precox, 2 cases of gross organic dementia, and 1 climacteric melancholiac. In 6 of these cases cholin was tested for, and found, in the nerve centres, and in 1 case also in the blood. A precipitate of *proteid* was obtained in all cases, the amount being large in 17 cases (especially in the general paralytics), and nearly always associated with the presence of cholin.

In the record of his 34 cases, Dr Coriat correlates under each the clinical picture, the analysis of the cerebro-spinal fluid, and the results of the post-mortem and histological examination which was made in many instances.

C. C. EASTERBROOK.

**ACUTE PARESIS WITH REPORT OF A CASE; THE CLINICAL (181) HISTORY AND PATHOLOGICAL FINDINGS** STEWART PATON and G. Y. RUSH, *Am. Journ. of Insan.*, Vol. lxx. No. 3, 1903.

**THE** case recorded is one of the *forme foudroyante* or galloping paresis. The authors consider that such cases are not so common

as are generally supposed, many so-called cases being but acute exacerbations of a previous condition which has been more or less latent. Only those cases which present the following features should, according to the authors, be classified under the *forme foudroyante*:—

1. Either the clinical history must be so complete as to render it possible to eliminate the occurrence of a prodromal period in which the symptoms are of a sub-acute or chronic type ; or,

2. If the patient dies during the period of acute delirious excitement it is essential that the pathologist should determine whether there is a definite organic lesion present sufficient to have caused death.

3. If the pathological findings indicate the existence in the central nervous system of a sub-acute or chronic process, the case cannot be included in this group. The deductions based upon the pathological findings are decisive even although the clinical history may suggest an acute onset and termination. The following case is of interest mainly histologically, the history of the patient during the month prior to his admission to the hospital being somewhat deficient.

The patient, a male aged 37, married, was admitted to hospital on October 29, 1901, and died March 2, 1902.

The history gives nothing worthy of note, although the patient in his emotional periods says that he contracted syphilis several years ago.

On admission the pupils were equal, dilated and reacted normally. Dermatographia was fairly well marked and the cremasteric reflexes present on both sides. The radial and triceps reflexes were active and the patellar reflex present. There was no ankle-clonus, Romberg's sign was not present. Voluntary motor impulses were weak. The patient continued more or less excited, being noisy, continually writing, and using a number of inarticulate expressions, until February 23, when he had nineteen convulsions. He remained semi-comatose with a slight general improvement on February 26, but died on March 2.

Notes from autopsy, which was performed two hours after death:—The skull-cap is not adherent ; no roughening of inner surface of skull or outer surface of dura. Patches of opacity of pia-arachnoid on inner and upper surfaces of hemispheres ; none over base. (No mention is made in the report of presence or absence of adhesion of pia-arachnoid to cortex.) The sections of dura from frontal region show small recent hæmorrhages.

The summary of general anatomical findings is given as:—Pachymeningitis hæmorrhagia ; chronic diffuse nephritis of mild degree ; marked fatty degeneration of the liver ; a small area of bronchitis due to the presence of an infected mass.

No less than six pages of print with two pages of very fine plates are devoted to the histology of the brain. From the author's resumé I take the following:—

"The pathological findings reveal nothing that is essentially characteristic of the disease. We have noted in particular a nerve-cell alteration which is in no sense pathognomonic" (pigmentation and chromatolysis being well marked). "There is a general and not a localised disappearance of fibres from all cortical areas. The neuroglia shows an increase of the cellular elements, particularly of the larger size spider cells, with but little, if any, increase of the fibres. The blood-vessels, particularly the larger arteries of the cortex, are well filled with blood, in some cases this over-filling extends to the smallest vessels; many round nuclei are found in the perivascular spaces. In most of the cortical areas, numbers of plasma cells are present. We have failed to find them in the tissue surrounding the vessels. The character of the change in the various organs as well as in the central nervous system suggests a general intoxication. The vascular changes of the cortex are not sufficient to account for the degeneration of the nerve elements."

R. G. WHITE.

**PUERPERAL INSANITY.** ROBERT JONES. Abstract of a paper read (182) before the Obstetrical Society of London, Jan. 7, 1903.

THE paper is based upon a personal experience of 259 cases of puerperal insanity, divided into — 120 cases during the actual puerperal period, 83 during lactation, and 56 during pregnancy.

Insanity is stated to occur once in every 700 confinements.

Insanity is stated to be of a characteristic form after confinement, amounting to an almost nosological entity; but this is not the case during pregnancy nor during lactation, there being no definite type of insanity occurring in connection with these two stages. The divisions are, however, more convenient than typical.

The following propositions are advanced by the author:

1. The insanity of pregnancy is more common in first confinements among single women, the disappointment, shame, and disgrace of illegitimacy being an important factor in the mental break-down.

2. During pregnancy the mental condition is more often acute melancholia than acute mania, and suicidal symptoms, which occurred in 41 per cent., have to be carefully guarded against.

3. The insanity of pregnancy is divided into that occurring during the early months and that occurring during the later months, and in these the nearer the insanity in point of time to the confinement, the more acute are the mental symptoms. Insanity is not more frequent when the sex of the child is male.

4. The insanity of the puerperium comes on after the first confinement in 33 per cent. of the cases, and supervenes suddenly rather than gradually.

5. The cases which occur during lactation present characters of marked general physical exhaustion, and mentally are more often of the depressed than of the maniacal form. Lactation insanity becomes chronic oftener than the insanity of the other periods. There is a tendency to low forms of inflammation, thrombosis, gangrene, and phthisis, during the insanity of lactation. Both suicidal and infanticidal promptings are more common in lactational than puerperal cases,—that is, in cases where insanity commenced more than six weeks after confinement.

6. The early symptoms of threatening insanity are loss of sleep and headache, and these should be a forewarning of mental break-down. The busy delirium of hallucinatory character, ending in acute restless, purposeless mania with religious and erotic delusions, is characteristic of this variety. The writer suggests a close analogy between the emotions of love and religion, and agrees with Simpson that the organ diseased gives a type to the insanity, and that in women suffering from affections of the generative organs the delusions are more likely to be connected with sexual matters.

7. *Ætiology*.—Heredity is more marked and in the direct maternal line in puerperal and lactational insanity, and is equally paternal and maternal in the insanity of pregnancy. A previous record of hysteria is frequent in puerperal insanity. The question of marriage of hysterical persons is considered.

8. The *pathology* is that of heredity and stress. Is the stress due to a toxin?

9. As regards *prognosis*, cases of insanity during early pregnancy improve towards the end of pregnancy, whereas those of late pregnancy become worse at the puerperium. Puerperal insanity is markedly recoverable. Improvement is rapid, being often complete in three months, but generally takes four to five months.

10. *Treatment*.—All cases presenting headache and sleeplessness must have absolute quiet and rest, and sleep must be procured. Home treatment in all cases if possible. Guard against unusual and sudden impulses of suicide and infanticide. The presence of the husband aggravates the symptoms. There is much necessity for a liberal and stimulating dietary. Change is necessary in puerperal insanity when cases tend to become stuporose. Menstruation is a sign of mental improvement. Purgatives and iron are well borne.

AUTHOR'S ABSTRACT.

**INSANITY FROM HASHEESH.** JOHN WARNOCK, *Journ. Ment. (183) Sc.*, Jan. 1903, p. 96.

THE author first gives some historical notes on the use of cannabis indica, then contrasts hasheeshism and alcoholism, and compares the Egyptian and Indian experience of the drug.

In Egypt hasheesh is consumed extensively, being employed, as tobacco, opium, etc., in other countries, for its euphoristic effect on the nervous system, but the better classes condemn its use.

The diagnosis of insanity from hasheesh depends on the history and the patient's statements.

The clinical types are six in number. 1. Temporary intoxication, rarely coming to asylums, is similar to that of opium and alcohol, showing a stimulant and sedative effect, drowsiness, pleasant exaltation, etc., with half-waking dreams. In this state the smoker may show irritability and loss of self-control; a staggering gait like alcoholism, and a dreamy state similar to that induced by opium. The mental pose is more "subjective" than that of the alcoholic, which is "objective," and less than that of the opium eater. 2. Delirium from hasheesh: Hallucinations of senses, delusions and exaltations, marked restlessness and insomnia; motor phenomena are absent, suicide is rare. This is a less grave state than the similar condition produced by alcohol, *i.e.* delirium tremens. 3. Mania: From a short mild excitement to a fatal furious mania; exaltation, grandiose and persecutory delusions, restlessness, incoherence and moral depravity; suicide rare, and hallucinations not so marked as in alcohol. Some cases are melancholic. 4. Chronic mania: Very similar to ordinary chronic mania but hallucinations are not so frequent and patient is happier than the morose and suspicious alcoholic. Suicide again rare. Motor and sensory troubles are absent. 5. Chronic Dementia is the final stage, similar to that of alcohol, but there are no motor or sensory symptoms. 6. "Cannabinomania," describes the condition between the attacks and is very similar in most respects to dipsomania. The differences are due to racial peculiarities, but the craving is not so marked and there are no phenomena of deprivation, so that the cessation of the habit is easier than in the case of alcohol or opium.

Contrasting insanities from hasheesh and alcohol, the author states that the following points stand out:—Suicide is comparatively rare in the former; hasheesh is a more important cause of insanity in Egypt than alcohol is in England, and it is as great a cause of crime; its use is not followed by characteristic anatomical lesions.

The Government prohibitions do not stop the use of hasheesh, but insanity from it is decreasing. The author thinks that if its

use were entirely prevented alcohol would replace it to the greater detriment of Egypt.

In India, *cannabis indica* is extensively used. The commission of 1894 report that its moderate use is not injurious. In 1892 it caused 7·3 per cent. of cases. In Egypt, Cairo Asylum, during 1896-1901, 27 per cent. of cases were attributed to it. In that country few females use hasheesh and it is noteworthy that insanity is there three times more common in males than in females, a very different state of affairs to that in England.

C. H. G. GOSTWYCK.

### TREATMENT.

**A CONTRIBUTION TO THE RADICAL CURE OF EXOPHTHALMIC (184) GOITRE, WITH ULTIMATE RESULTS IN EIGHT CASES TREATED BY THYROIDECTOMY.** J. ARTHUR BOOTH, *Journ. Nerv. and Ment. Dis.*, Sept. 1902.

THE author gives a triple classification of cases of exophthalmic goitre, based upon the different groups of symptoms observed. (1) cases dependent on changes in the central nervous system; (2) cases due to disease of the cervical sympathetic; (3) cases arising from excessive or altered function of the thyroid gland. In cases of the first class no benefit is to be derived from operative measures. In cases of the second variety the disease has been found, by post-mortem examinations, to be most often situated in the inferior cervical ganglion. Rehn, of Frankfort, in 32 cases of resection of the sympathetic, reports as follows: 28 $\frac{1}{6}$  per cent. are cured; 50 per cent. improved; 12 $\frac{1}{2}$  per cent. unimproved; and 9 $\frac{3}{10}$  per cent. deaths. Jennesco, in 15 cases, reports 6 cured, 4 improved; in 5, sufficient time had not elapsed to give results. There were no deaths.

In the third class we have positive proof of changes in the thyroid in a very large number of the instances. Schultze and Rehn report that in 319 cases, in which the thyroid was operated upon, 175 were cured (51 per cent.), 89 improved (28 per cent.), 13 unimproved (4 per cent.), 41 died (13 per cent.). In the clinic of Mikulicz 18 patients were operated on. 1 died of secondary hæmorrhage, 10 were permanently cured and 7 were improved. Kummel, of Hamburg, gives the following results in 177 patients: 57·6 per cent. cured; 26·5 per cent. improved; 2·3 per cent. unchanged; 13·6 per cent. died. The author gives details of 8 cases of his own. One died after operation, but this was probably due to uremia as the patient had diabetes and the urine contained a large amount of albumen, and hyaline and granular casts. All were females. In 7, enlargement of the thyroid was noticed first.

In 6, exophthalmos was present and was improved in 3 and disappeared in 3. In all the operation was a partial thyroidectomy. Symptoms such as tremors, rapid pulse, palpitation, and excessive sweating, were present in each case. The results were: 6 permanently cured, 1 improved, 1 dead. From records it appears that in cases of operations on the sympathetic sudden deaths are less numerous, and relief from exophthalmos more common than with operations on the thyroid.

The author comes to the following conclusions:

(1) Cases of Graves's disease may be completely cured both by thyroidectomy and bilateral section of the sympathetic.

(2) In view of the fact that some cases are cured by internal medication, there must be a certain proportion in which the affection does not induce structural changes in any organ.

(3) No theory can be regarded as adequate without taking into consideration the function of the thyroid gland.

(4) Three factors must be considered in the production of the disease: (a) The central nervous system; (b) the connecting fibres, sympathetic and vagus; (c) the thyroid gland.

(5) A lesion in one of these parts may produce a specific alteration in the others, the consequences of which, together with the exciting cause, may give rise to the symptoms of Graves's disease.

A. B. Ross.

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## Review

### THE HISTOLOGICAL BASIS OF AMENTIA AND DEMENTIA.

By JOSEPH SHAW BOLTON, *Archives of Neurology*, from the Pathological Laboratory of the London County Asylum, Claybury, Essex, vol. ii., 1903.

THIS paper, which extends to nearly 200 pages, "contains the results of a research on the physical basis of mental disease, the method used being that of micrometric examination of the cortex cerebri." The author explains that "in order that suitable cases might be selected for the purpose of study, it was first necessary to determine whether any constant relationship existed between the morbid appearances found in many cases of mental disease and the clinical types of insanity manifested by the patient." The first and larger part of his paper consists of an account of this preliminary investigation, which was successful in its object, and which embraced "the clinical and pathological study of 200 cases of mental disease (excluding dementia paralytica) which appeared consecutively in the Claybury Laboratory." To this is added, however, "an account of the etiology and pathology of dementia

paralytica, which is necessarily introduced in order to demonstrate the relationship which, in the opinion of the writer, exists between this and other varieties of mental disease."

Part I. is entitled "The Morbid Anatomy of Dementia, together with certain observations on the General Pathology of Mental Disease." The clinical types between which and certain morbid appearances he claims to have found a constant relationship consist in various degrees of dementia. He classifies his cases in five groups, namely, "Cases without appreciable dementia," "Cases with appreciable dementia," "Cases of insanity with moderate dementia," "Cases of severe dementia which still showed symptoms of insanity," and "Cases of gross dementia." He gives in tabular form a general summary, in percentages, of certain of the morbid appearances observed in the 200 cases. He finds that the morbid appearances inside the skull-cap "vary directly with the amount of dementia present." "They are," he says, "the microscopic equivalents of, and vary in degree with, the amount of dementia present, and are otherwise independent of the duration of the insanity." The morbid appearances referred to consist in a thickened or adherent condition of the dura, subdural deposit, subdural excess, the readiness with which the pia-arachnoid strips, subarachnoid excess, dilated and granular conditions of the lateral ventricles and granular lateral sacs of fourth ventricle. He adds some notes upon the weights of the pia-arachnoid and cerebral hemispheres and upon degeneration of the cerebral vessels. There follow sections upon "The Pathology of Dementia," "The Pathology of sub-dural Deposits," "The effect of Gravity on the intracranial contents of the Cadaver," "The influence of Heredity on the development of Mental Disease," "The General Pathology of Mental Disease," and "Dementia Paralytica." He considers that "neuronic insufficiency (as regards development, durability, or both) is the necessary antecedent of mental disease." He would appear to include all forms of insanity in two groups, namely, "Amentia" and "Dementia." The former term denotes "the mental condition of patients suffering from deficient neuronic development," the latter "the mental condition of patients who suffer from a permanent psychic disability due to neuronic degeneration following insufficient durability." He attributes some importance to "stress" as a determining cause of the time of onset of an attack of insanity, but maintains that the influence of this factor in overcoming the durability of insufficient neurons, even of the highest types, is limited, and that the dementia is very rarely more than moderate in degree unless one of two additional factors supervenes, namely, "ordinary vascular degeneration" and "neuroglial and vascular reparative proliferation which may follow syphilitic infection, and which continues to increase, *pari passu*,



with neuronie degeneration, till gross dementia and death ensue." He holds that "dementia paralytica is not an organic disease of the brain, but is a branch of ordinary mental disease, special clinical and pathological features being introduced in consequence of a former attack of syphilis ; and that it develops solely in the actual or potential subjects of those types of mental disease which, owing to a hereditarily deficient durability of the cortical neurones, tend to end in dementia."

Part II. has the title of the paper. It contains some additional remarks upon the author's "Classification of Mental Diseases," and an account of his researches upon "The General Histology of the Cerebral Cortex." The latter includes a statement of the results of the cortical measurements made in twenty cases. The author finally summarises his conclusions regarding the functions of the primary cell layers of the cortex. The broad generalisations that he bases upon his observations have already been stated.

There are several features of this paper that seem to call for critical remark here. Perhaps its most striking feature is the almost complete lack of collation of the observations recorded with those of other workers. A writer in any department of progressive science may state that circumstances "have compelled him to limit himself to the recording and classifying of his own observations," but he does not thereby alter the fact that his observations can have no precise value until they are given their proper place, if they have any, in the slowly rising edifice to which the labours of others have already contributed. It is one of the recognised rules of the scientific method of research that an investigator should, as far as possible, make this adjustment himself. If he does not, he is almost certain to fall into many serious errors, and to mislead when he is trusted to guide aright. To many readers of the *Archives of Neurology*, Dr Bolton's descriptions of pathological changes, given in Part I., will have the appearance of important original observations, whereas, with a few trivial exceptions, they really concern facts with which every asylum pathologist is familiar, and which are already recorded in literature. Instances of a similar kind are not wanting in Part II. For example, he states the conclusion that the prefrontal lobes are "the part of the cerebrum concerned with the highest function of mind, namely, attention and general orderly co-ordination of psychic processes," as a deduction from his own observations. No doubt he will claim that it is, but he gives the reader no hint that the question is a very old and still highly controversial one, possessing a voluminous literature. His "general pathology of mental disease," based upon the fundamental idea that neuronie insufficiency is the necessary antecedent of mental disease, amounts to one more attempt to create a pathology of insanity which is inde-

pendent of general pathology. The day has passed in which such attempts can have any utility. They even do harm by obscuring the paths that lead to important therapeutic and preventive measures.

A word also requires to be said about the author's attempt to establish an exact correlation between the degree of dementia, on the one hand, and the thickness of certain portions of the cortex and "morbid appearances inside the skull-cap," on the other. It is not permissible to institute a comparison, except in the most general terms, between phenomena that are of entirely different orders and that have no characters in common. Dementia cannot be weighed, or measured in micromillimetres. It is also to be observed that before Dr Bolton wrote about the subject, several observers had pointed out that in cases of severe secondary dementia there is a demonstrable paucity of nerve-cells in the cerebral cortex dependent upon complete destruction of a considerable proportion of these tissue-elements during the previous attack of acute insanity. Of course, accompanying this loss of tissue there is a narrowing of the cortex. These are the essential facts. Dr Bolton, in this publication, as well as in his paper on the subject in the *Journal of Mental Science*, makes no reference to these prior observations. He did not originate this conception of the narrowing of the cortex in dementia; he has only elaborated it. The really original and valuable part of his work is that which concerns the exact measurement of the cortex, the comparison of brain with brain, and the localisation of the special seats of under-development and loss of nerve-cells in idiocy and secondary dementia respectively. It may be permitted to hope that he will continue this important line of research, for which he is undoubtedly well equipped.

The section upon "The effect of gravity on the intra-cranial contents of the cadaver" resolves itself into an attack upon certain published views of my own. Dr Bolton says: "At death a rapid change occurs in the position of the blood contained in the cranium, that in the arteries decreasing in quantity and that in the veins increasing, the total amount being approximately or exactly the same. . . . That this blood exchange actually does occur will be clear from the experiments contained in the present section. That, as suggested by Hill, a considerable amount of blood is held up in this way in the veins and sinuses of the cranium, which would escape on opening the skull-cap in a position in which gravity can act, seems hardly to require proof in view of the important researches which have during the past few years been carried out by this painstaking and experienced physiologist. In view, however, of the opinions expressed in the recent work by Ford Robertson on the 'Pathology of Mental Diseases,' this is not the

case, and the writer of the present paper has consequently put the question to experimental proof." If any clear meaning can be attached to this passage, it is that I have denied (1) that when the heart's action ceases and the arterial blood pressure falls to *nil*, the amount of blood in the cerebral arteries diminishes, while that in the cerebral and dural veins increases; and (2) that when the calvarium is sawn across at a post-mortem upon the human subject, a certain amount of fluid drains away from the solid intracranial contents. Now the fact is I have never traversed these statements. On the contrary, the occurrence of the first phenomenon is recognised in the very chapter to which Dr Bolton refers; that of the second is too obvious to have required mention. It may be observed that the fluid that drains away consists partly of cerebro-spinal fluid, and partly of blood or serum contained especially in the dural sinuses. The passage that Dr Bolton quotes as "the statement referred to" occurs in the course of a discussion of the value to be attached to post-mortem evidence of cerebral congestion, and does not relate to the *Monro-Kellie* doctrine as he unfortunately implies. The particular question with which this passage deals is whether or not Hill was right in affirming that "the whole blood-content of the brain may change at the moment the pathologist opens the skull" in accordance with the direction in which gravity is permitted to act. I say—"I scarcely think that this statement is in accord with the general experience of pathologists. I have never myself observed evidence of such changes occurring to any important extent. It has not been my experience that, on opening the skull and reflecting the dura, an anæmic brain can be made a congested one by lowering the head of the cadaver, or that a congested brain can be made anæmic by raising it above the level of the trunk. It would indeed be surprising if such extreme and rapid alterations could occur, for by the time that autopsies are commonly made the blood in the vessels has generally undergone more or less extensive clotting." It must be obvious to the reader that this is a point essentially different from the two others already defined. Dr Bolton argues as if they were the same. Utter confusion is the inevitable result. The experiments that he has been at the trouble to make have no bearing upon this third point. He made trephine holes in the most elevated portion of the skull and poured water through them after incising the dura. He naturally found that there was evidence that a certain diminution occurred in the volume of the contents of the intracranial cavity when air was admitted. He assumes that only blood has been displaced. It is certain that also cerebro-spinal fluid has drained away. In any case the experiments do not prove that under the conditions that commonly attend post-mortem examinations, a congested brain can become an anæmic

one if the calvarium is removed whilst the head is elevated, or that an anæmic brain can appear as a congested one if the calvarium is removed whilst the head is dependent. These are the only points in question in the passage he quotes from me.

Dr Bolton states that his experiments "indirectly afford the strongest support to the Monro-Kellie doctrine." No one who understands the points at issue between the defenders of this doctrine and those who support the doctrine of Burrows, could make such a claim. The doctrine of Burrows recognises that the intracranial cavity is "closed," and that therefore the volume of the intracranial contents must be constant. It only differs from the Monro-Kellie doctrine in asserting that the volume of blood in the brain can vary in an important degree. My argument against the Monro-Kellie doctrine and in support of that of Burrows rests on the contention that the most able defender of the former has fallen into certain errors which, when corrected, leave the latter as the only one that is tenable. These errors consist, briefly, in this, that he has failed to take into account the variability of the quantity of fluid in the lymph spaces of the brain; and that, neglecting this fluid and being unaware of the great elasticity of all the tissues composing the brain (he describes it as "a viscous, inert mass of the consistency of a lump of putty"), he has accepted as an axiom what is really one of the most transparent of fallacies, namely, the doctrine of the incompressibility of the brain.

W. FORD ROBERTSON.

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# Review of Neurology and Psychiatry

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## Original Articles

### **BACTERIOLOGICAL INVESTIGATIONS INTO THE PATHOLOGY OF GENERAL PARALYSIS OF THE INSANE.**

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Asylums.)

Two years ago, Dr Lewis C. Bruce (1) and one of us (2) published separate papers, in which it was contended that the toxæmia of general paralysis of the insane is of gastrointestinal and bacterial origin.

In the first of these papers, this contention was based upon clinical observations, in which were included a study of the occurrence of leucocytosis in a series of cases and of the agglutinative power of the blood serum upon the bacillus coli. In the second it was founded upon the results of histological investigations into the changes in the stomach, intestines and liver, as well as upon certain previously recorded observations. It was also maintained in the latter paper that the part played by syphilis (and other recognised causes of general paralysis, such as the prolonged action of alcohol and of lead salts) is essentially that of altering the natural immunity. It was further concluded that there is a partial breakdown of those forces by which the harmful development of the micro-organisms that constitute the ordinary flora of the alimentary tract is normally

prevented, but that the specially injurious toxins are probably the products of only a few bacterial forms, among which some of those of the colon group may have a special importance. In a later paper (3) it was stated that bacteria can be shown to have obtained a lodgment among the epithelial cells of the gastric or intestinal mucous membrane in this disease.

These investigations, whilst bringing forward a considerable amount of clinical and histological evidence of the occurrence of severe toxic infection from the alimentary tract, left the exact nature of the alleged bacterial attack a matter of conjecture, and gave no indication for experimental investigation, by which alone, it seemed probable, the value of the hypothesis could be definitely determined. It seemed to us that the question might be further elucidated by the employment of bacteriological methods. We therefore began, more than a year ago, at the Royal Edinburgh Asylum, a research of this kind, the general results of which have already been stated in a preliminary note (4). Our original object was to ascertain if cases of general paralysis have any distinctive features in the bacterial flora of the alimentary tract, and if micro-organisms are commonly present in the blood and internal organs. In the second case from which we had an opportunity of making cultures post-mortem, we obtained an organism resembling the Klebs-Löffler bacillus from the sub-mucosa of an inflamed part of the ileum, from a pneumonic lung and from the brain. Our attention was therefore at an early stage of the research directed to this organism. As the immediately succeeding cases also presented clear evidence of the prominence of the same bacillus, our investigation became narrowed down to an enquiry as to its presence in various situations in individual cases, for it appeared to us that if this organism was constantly present in the alimentary tract, and especially if it could be isolated from internal organs, we had a sufficient indication for an experimental investigation, without seeking to elucidate further the special characters of the bacterial flora of the alimentary tract. This experimental investigation was kindly undertaken for us by Dr Theodore Shennan, and whilst we awaited its results, we endeavoured to obtain further information regarding the occurrence of the bacillus in cases of general paralysis. As has been stated in the second of the two preliminary notes (5) that have been published, the experimental

evidence shows that this organism is capable of producing a fatal disease with nervous symptoms in rats, and that the anatomical changes produced have a distinct resemblance to those that occur in dementia paralytica.

#### PREVIOUS BACTERIOLOGICAL INVESTIGATIONS IN CASES OF GENERAL PARALYSIS.

D'Abundo (6), in a paper published in 1889, stated that he had observed various common micro-organisms in the blood of general paralytics. He attributed their presence to the occurrence of bed-sores, but in a later paper expressed the opinion that they had entered by way of the bladder. Piccinino (7) in 1894 recorded some very similar observations. Two years later (8) he described the occurrence, in the brain of general paralytics, of an organism having the morphological characters and staining reactions of the bacillus described by Lustgarten in syphilis. Grimaldi (9) made cultures from the blood and urine in ten cases. He obtained a streptococcus from the blood in one case, and the colon bacillus from the urine in four; the other observations were negative. He thought that the frequent presence of the latter organism in the urine was dependent upon disorder of the alimentary tract. He seems to have been the first to maintain that the intoxication of general paralysis is of gastro-intestinal origin. Montesano and Montessori (10) in 1897 recorded the results of a very complete bacteriological examination of the cerebro-spinal fluid, obtained by lumbar puncture from eleven cases of general paralysis. They could not find any micro-organisms by microscopical examination of the deposit obtained by centrifuging the fluid. Direct inoculation of the fluid into guinea-pigs gave negative results in eight cases, certain characteristic morbid phenomena, with death on the fourth day, in two cases, and death from tetanus in the remaining one. They found a special bacillus, a variety of the *bacillus viscosus*, in the first two fatal cases, but could only find a streptococcus in the animal that succumbed to tetanus. Cultures made upon nutrient media were positive in eight cases; three cases were negative. Among the organisms obtained were streptococci (thrice), staphylococci, sarcinæ and the *bacillus viscosus* (four times). The tetanus bacillus was obtained from

the blood of the case, the cerebro-spinal fluid from which caused tetanus in the guinea-pig; but only streptococci were isolated from this fluid. They stated that the *bacillus viscosus* was not identifiable with any described in literature, but admitted that it might be a variety of the *bacillus coli*, though it was immobile, and retained the stain in Gram's method. It gave no indol reaction. It was extremely virulent to guinea-pigs and rabbits.

Although very numerous observations have since been recorded upon the cytological and chemical characters of the cerebro-spinal fluid taken from general paralytics by means of lumbar puncture, no one, as far as we have been able to ascertain, has endeavoured to follow up the line of research initiated by these two Italian observers.

#### AUTHORS' INVESTIGATIONS.

Our investigations upon cases of general paralysis have comprised enquiries regarding—

- (1) The presence of micro-organisms in the blood during life.
- (2) The special characters of the bacterial flora of the alimentary and respiratory tracts and the presence of micro-organisms in the tissues at post-mortem examinations obtained under as favourable conditions as possible.
- (3) The presence of the Klebs-Löffler bacillus in the mouth and throat and in the expectoration, during life.
- (4) Any possible specific agglutinative action of the blood serum upon cultures of the Klebs-Löffler bacillus; and
- (5) The action of anti-diphtheritic serum upon the progress of the disease.

#### (1) *Cultures from the blood during life.*

Cultures upon various media were made from the blood of six cases by the following method. Either the point of a finger or the lobe of an ear was first thoroughly cleansed with soap and water and dried. A little absolute alcohol was next poured on and allowed to evaporate. A drop of hot sterilised vaseline was then applied with a brush, which, being fixed on the inner side

of the cork of the tube containing the vaseline, had been sterilised along with it. The part was then pricked with a sterile needle. From the large drop of blood which immediately rose upon the surface of the vaseline, loopfuls were quickly taken with a platinum needle and smeared over the surface of the culture media. Stab cultures were also made. We believe that by this procedure it is possible to avoid contamination by the organisms that are normally present in the superficial layers of the skin, because the hot and fluid vaseline soaks into the dry epithelium and then solidifies, so that the needle forms for the blood a tubular channel lined by a sterile and impenetrable medium.

In all of these six observations the culture media remained sterile. The clinical states of the patients were briefly as follows: (1) Initial stage of congestive attack; (2) slight congestive attack; (3) acute maniacal excitement, disease rapidly progressive; (4) severe congestive attack, death two days later; (5) congestive attack; (6) congestive attack, death on following day.

In three other cases, 5 c.c. of blood were taken from a vein of the arm and mixed with 30 c.c. of byno-hæmoglobin broth. Cultures were made from this after forty-eight hours' incubation at 37° C. In two cases there was no growth, in the third a staphylococcus appeared. The first case was that of a woman in a congestive attack which resulted in death two days later; the second was that of a man in the third stage, and the remaining subject was a man also in the third stage who had just passed through a congestive attack. The presence of the staphylococcus, which occurred in the cultures from the last case, was almost certainly due to an accidental skin contamination which was noticed at the time of the operation.

These observations lead us to conclude that the blood of general paralytics is as a rule sterile, although they do not of course exclude either the possibility of the occasional entrance of a few organisms into the blood as in many other diseases, or the phenomenon of terminal invasion by bacteria.

#### *(2) Cultures from cases examined after death.*

In the earlier part of our researches we employed as culture media, chiefly ordinary agar, glucose agar, nutrient gelatin and

Löffler's serum, but later we made use of byno-hæmoglobin agar, a medium we devised as a substitute for Löffler blood-serum and which we have found can be depended upon to show the Klebs-Löffler bacillus in the form in which its metachromatic granules are prominent.

The following is the composition of this medium :

Byno-hæmoglobin (Allen and Hanburys)	25 c.c.
Agar . . . . .	10 grammes
1 per cent. Caustic Soda . . . . .	15 c.c.
Water . . . . .	500 c.c.

These ingredients are placed in a litre flask, or other suitable receptacle, and kept at a temperature of 100° C. for about two hours. The medium cannot be prepared in the autoclave (at least if an ordinary litre flask is used) owing to the large amount of frothing that occurs. It is essential that after the temperature has passed 60°, and until the boiling point is reached, the medium should be stirred or agitated in order to prevent the formation of large coagula. The medium may be filtered in the usual way. It has then a faint amber colour, but otherwise the appearance of an ordinary agar medium. We prefer, however, to use it without filtering, as the opaque brown appearance which it then presents is much more suitable for the study of surface growths. After sterilisation the medium is poured into suitable tubes, which are sloped until solidification takes place. If contamination at this stage is feared, the tubes should be further sterilised in the dry chamber of a water bath at a temperature not exceeding 95° C. for two to three hours on two successive days. Whether this last precaution is taken or not, the tubes should always be incubated for two or three days before use in order to insure that they are sterile. Although this medium may be trusted to show the Klebs-Löffler bacillus in the form in which its metachromatic granules are prominent, it has the disadvantage that the organism does not live upon it for more than from three to six days. It is therefore necessary to make subcultures every second or third day.

We have used the ordinary staining methods and the special method of Neisser for the Klebs-Löffler bacillus.

After reading the paper of Beaton, Foord Caiger and Pakes (11) upon the value of Neisser's stain in the diagnosis of diph-



theria, we employed their modification with, we think, very decided advantage. This modification consists simply in allowing the acid methylene blue and vesuvin brown stains to act each for two minutes, instead of for the few seconds prescribed by Neisser.

Cultures were made from the submucous or mucous coat of the small intestine and stomach, after searing the outer surface and incising the peritoneal and muscular coats with a sterile knife, from the inner surface of these organs, from the brain (by puncture with a straight platinum needle immediately on raising the dura) and other internal organs.

When we found that the Klebs-Löffler bacillus appeared in successive cases in one or other of the tubes inoculated from these situations, we made cultures also from the tonsils and pharynx, and, at a still later stage, from the bronchi. Stroke cultures were chiefly made and the examinations were begun on the following day. Subcultures were in several instances made from the inoculated media within six or nine hours, as was suggested to us by Dr Shennan. We have found this procedure of considerable assistance in the isolation of the bacilli. We have, however, frequently obtained pure colonies from the first cultures.

We have examined altogether twenty cases. The Klebs-Löffler bacillus was obtained from seventeen. In the three from which it was not isolated, it was readily found in microscopic preparations of the stomach in two of the cases and in a film preparation of the material on the surface of the trachea in the third. The following table shows the number of times the organism was isolated from different situations :

Inflamed portion of ileum,	5 cases out of	16
Stomach . . .	7	16
Tonsils or pharynx . .	9	12
Bronchi . . .	3	5
Lung tissue . . .	3	6
Brain . . .	4	16

The tonsils were almost constantly found to contain small purulent foci on section, and it was chiefly from these that the growths of the diphtheria organism were obtained. The bacillus was not isolated from the bone-marrow (five cases), from the heart's blood (two cases) or from thrombosed pial veins (two cases). Out of

the four cases in which the organism was obtained from the brain, it was associated in the first with the bacillus coli, in the second with streptococci, in the third with bacillus coli and staphylococci, whilst in the fourth it occurred alone. Of the twelve other cases in which the Klebs-Löffler bacillus was not obtained from the brain, ten gave no growth, whilst the remaining two yielded pure cultures of the bacillus coli.

Six cases of other forms of insanity were examined by the same methods. The Klebs-Löffler bacillus was obtained from two. One of these was clinically a case of syphilitic insanity with organic disease of the brain. The organism appeared in a culture from an inflamed patch in the ileum. The other case was one of chronic melancholia with extensive tubercular disease of vertebrae, ribs, etc. In this case the organism was obtained from the tonsil.

Regarding the cultural and morphological characters of this organism which appears to have special prominence in cases of general paralysis, it is impossible for the present to say more than that they are those of the Klebs-Löffler bacillus (see Plate 5). Its action upon glucose broth has been tested in several instances and the rapid formation of acid has been clearly manifested. The organism is therefore not Hofmann's bacillus.

(3) *The presence of the Klebs-Löffler bacillus in the mouth and throat and in the expectoration during life.*

We have succeeded in obtaining cultures of the Klebs-Löffler bacillus from the tonsils, pharynx, carious teeth and saliva of cases of general paralysis during life, and have also observed the organism in considerable numbers in film preparations made directly from material from these surfaces. In the only instance in which we have been able to obtain expectoration for examination, the Klebs-Löffler bacillus was found to be present in enormous numbers. In many other cases we have failed, with the limited time at our disposal, to obtain evidence of the presence of this organism in the situations mentioned. There are many difficulties attending such investigation, chief among which is the great abundance and variety of the bacteria present in these cases. We propose to deal with this subject in a later paper when we have had opportunities of making further observations.

FIG. 1.



FIG. 1.—Stroke culture of Klebs-Löffler bacillus isolated from a case of general paralysis. Byno-haemoglobin agar; 24 hours' growth.

FIG. 3.

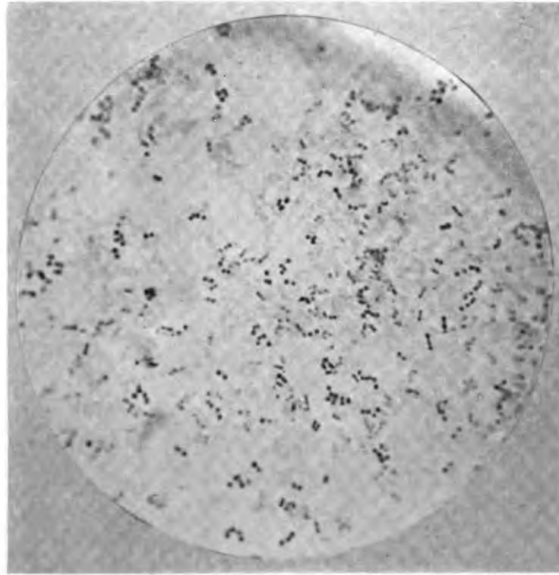


FIG. 3.—Klebs-Löffler bacilli from a first culture (24 hours' growth) from the surface of the stomach of a case of general paralysis. Glycerine blood-serum. Neisser's staining method ( $\times 1000$ ).

FIG. 4.



FIG. 4.—Klebs-Löffler bacilli from the fourth sub-culture from the tonsil of a case of general paralysis. Byno-haemoglobin agar. Neisser's staining method ( $\times 1000$ ).

FIG. 2.—Stroke culture of same organism, showing isolated colonies. Byno-haemoglobin agar; 48 hours' growth.



(4) *Attempts to obtain a specific agglutinative action of the blood-serum upon cultures of the Klebs-Löffler bacillus.*

We have used the methods employed in the serum diagnosis of typhoid. In normal blood-serum mixed with an equal quantity of a broth culture of the organism we have isolated, only imperfect agglutinative action can be observed. We have not found any distinct evidence of more energetic agglutinative action on the part of the blood-serum of general paralytics. It is to be borne in mind that an agglutinative test is of no value in the diagnosis of acute diphtheria, and that in general such phenomena are only clearly demonstrable in the case of flagellated organisms.

(5) *Observations with anti-diphtheritic serum in cases of general paralysis.*

Observations upon the effect of anti-diphtheritic serum were made in six cases.

The first case was that of a woman in the second stage of the disease, who, for six weeks prior to the commencement of the observation, showed an oscillating temperature with a distinct weekly curve between 97° and 101° F. During the following six weeks anti-diphtheritic serum, in doses of from 1000 to 2000 units, was injected every tenth day. Throughout this period and for three weeks afterwards the temperature kept below normal, the difference between the morning and evening temperature being seldom more than 1° F. Subsequently, for a period of sixteen weeks, the temperature presented the same character as that before the serum was used. There was no appreciable change in the other bodily symptoms or in the mental state.

Three male general paralytics in the second stage were treated with several injections, beginning with a dose of 2000 units and continuing with doses of 500 units. No effects were discerned which could be attributed to the action of the serum. Two of these cases are at present in a state of remission, the third is slowly progressive.

Another case was that of a man in the third stage, who had previously had two or three severe apoplectiform seizures, characterised by intense torpor, which passed off in the course of a few days. Serum treatment was commenced some hours after the

onset of a similar seizure, 2000 units being injected. The temperature fell steadily from  $101.4^{\circ}$  to  $97.4^{\circ}$  within twelve hours, and the patient regained consciousness, becoming very emotional and excited. There were subsequent distinct rises of temperature of short duration and two slight convulsive seizures, but after the fifth daily injection the temperature sank below normal and remained so for many weeks. The patient's mental condition distinctly improved for a time, a fact which was quite independently remarked upon by his wife. It is of interest to note also that in this case, during the period the serum was being used, the patient was subject to attacks of intense emotionalism and excitement, with general muscular rigidity, symptoms which he had not manifested when recovering from his previous seizures. After two weeks the injections were stopped as the patient continued to become weaker and more emaciated. Death occurred about two months later.

The last case was that of a woman, in the early third stage, who had for some time exhibited a confused mental state and irregular rises of temperature. She was confined to bed and had to be fed by hand. She was given 2000 units of serum every second or third day for a period of five weeks. After two injections she became more lucid than she had been for many weeks previously and replied intelligently when spoken to. After the third injection she was fit to be got out of bed, fed herself and actually employed herself in knitting. Subsequent injections seemed to produce bouts of maniacal excitement and resistiveness, similar to those observed in the previous case. During the treatment the temperature maintained a lower level than previously and the diurnal variation was less. The patient, however, subsequently relapsed into her former state and therefore, after the period mentioned, the treatment was stopped. She thereafter became progressively weaker and died about a month later.

#### REMARKS AND CONCLUSIONS.

It is now clearly established by the observations of many bacteriologists that the Klebs-Löffler bacillus may be present in the throat and on other mucous surfaces, sometimes in a very virulent form, in diseases which have not the clinical characters of acute diphtheria, and even in healthy subjects. Thus R. and

O. Neumann (12) found virulent diphtheria bacilli in some cases that were clinically merely cold in head. Symes (13) found the organism in most cases of atrophic rhinitis he examined. In 2 cases in which it was tested on guinea-pigs it was found to be virulent. Pearce (14) found this bacillus in one case of malignant endocarditis, in 19 cases of broncho-pneumonia, in 16 cases of middle-ear disease, in 8 cases of inflammation of the antrum of Highmore, as well as in many other non-diphtheritic morbid conditions. Park (15) found virulent diphtheria bacilli in about 1% of healthy throats examined in New York. Diphtheria was prevalent in the city at the time. It is also well known that persons attending upon cases of acute diphtheria very commonly have virulent Klebs-Löffler bacilli in their throats.

These facts might be regarded as proving that the constant presence of the diphtheria bacillus in the respiratory and alimentary tracts of general paralytics can have no etiological significance. There are, however, other considerations which bear upon the question and give it a very different colour.

Süsswein (16) has recently studied the fate of the Klebs-Löffler bacillus in the alimentary tract in cases of acute diphtheria. In 15 cases examined during life, he was unable in a single instance to detect the organism in either the gastric or intestinal contents. In 8 cases examined after death he was able to observe the bacillus in cover-glass preparations of the stomach contents in 4, but could obtain cultures in only 2.

We obtained the organism from the stomach in 7 out of 16 cases of general paralysis, and from the ileum in 5 out of the same number of observations. It would therefore appear that the organism is more readily isolated from the gastro-intestinal tract of general paralytics than from that of cases of acute diphtheria.

Another fact indicating that this bacillus has a pathogenic action in general paralysis is that we were able to obtain it from the brain in 4 cases out of 16 examined. Although we were unable to get a growth of the organism in 9 other inoculations which were virtually post-mortem blood examinations, we think that, taking into account all the facts bearing upon the point, we are only warranted in regarding its presence in the brain in these cases as evidence of a terminal general blood infection.

We cannot exclude the possibility of a local cerebral infection, but with the evidence we have this seems the less probable explanation. Whichever view is the right one, the presence of the bacillus in this situation constitutes very strong presumptive evidence that the organism has been exerting a pathogenic action upon the host. In this connection we would refer to a case described last year by Drs Johnson and Goodall (17) of Carmarthen Asylum, in which the diphtheria bacillus was isolated from the heart's blood. The case was one of "acute mental disorder, probably of toxic origin," thought to be secondary to influenza. It presented certain features which suggested dementia paralytica, although there was nothing in the appearances observed at the post-mortem to confirm this diagnosis. In an appendix to the paper, however, it is mentioned that "the tangential fibres and the superradiary were found to be scanty and degenerate." After studying the very clear account that the authors give of the case, and with the addition of this supplementary note, it seems to us that the case was almost certainly one of rapid general paralysis. We have occasionally seen cases of the kind in which a diagnosis could not be made from the naked eye appearances of the brain, and in which the true nature of the case was only determined by microscopic examination of the cortex.

Still another fact which supports the view that the presence of the Klebs-Löffler bacillus in cases of general paralysis has an important significance, is that it may be shown that in some cases the organism occurs in the bronchi or alimentary tract in very large numbers. In one of our cases the bacilli are so abundant in the lower respiratory tract that in a transverse section of one of the bronchi stained by Gram's method, in which microscopic examination proves that only these organisms have retained the stain, a broad violet ring lying on the surface of the mucous membrane can be seen with the unaided eye.

Another difficulty which some may find in the way of attaching importance to the presence of the Klebs-Löffler bacillus in cases of general paralysis, arises from the existence of what has been called "the pseudo-diphtheria bacillus." Without entering fully into the question here, we would merely say that the highest authorities are now agreed that, excluding the common Hofmann's bacillus, and perhaps one or two other organisms of rare occurrence, the "pseudo-diphtheria bacillus"



is merely an attenuated, non-virulent form of the organism which causes acute diphtheria. The Klebs-Löffler bacillus is obviously an organism that varies greatly in virulence under the influence of conditions that are not yet fully understood. Experimental evidence has already shown that some, at least, of these organisms which we have isolated from cases of general paralysis are not fully attenuated Klebs-Löffler bacilli, but that they have a certain modified virulence.

The recent great advances that have taken place in the knowledge of immunity have made it clear that it is impossible to understand the pathogenesis of any infective disease, and more especially of a chronic infective disease, apart from the consideration of the action of the defensive mechanisms. In a great many instances it is essentially the impairment of these defensive mechanisms, and not merely the presence of the micro-organism, that determines its rapid multiplication and assumption of a pathogenic action. There are good grounds for believing that the natural power of resistance against the attack of micro-organisms is lowered by the action of syphilis, alcohol, lead salts, excessive meat diet, and many other alleged causes of general paralysis. We think, however, that it is certain that there is a no less important impairment of the local defences in the alimentary or respiratory tracts, which may permit the permanent local lodgment and gradual heightening of virulence of an organism like the Klebs-Löffler bacillus. Thus we have observed one case in which there was a small, deep ulcer on the under aspect of the tongue, evidently of long standing. It may have been of gummatous origin, but this view was not borne out by microscopic examination. The cavity of the ulcer was filled with micro-organisms, among which there was an abundant number of Klebs-Löffler bacilli. Many general paralytics have extremely carious teeth; from some of these we have been able to isolate the same organism. In almost every case in which we have examined the tonsils of general paralytics after death, we have found, on section, small cavities containing pus, from which we have frequently been able to obtain a growth of the bacillus. It is easy to understand how necrotic foci, such as these we have instanced, may serve to lodge the organism for years, and act as a source from which it is continually being disseminated throughout the alimentary tract. It may, however, be unnecessary to

suppose the existence of such local foci, for the chronic bronchitis, which would appear to be an almost constant condition in dementia paralytica, may be sufficient, together with certain defects in the general power of resistance, to allow the bacillus to obtain a permanent hold upon the host. It is quite consistent with known facts regarding the variations in the virulence of the Klebs-Löffler bacillus, that under such conditions of existence, its pathogenic power may be gradually increased, either simply by long residence in the host, or by association with other organisms. There is abundant evidence that streptococci, staphylococci, varieties of bacillus coli, and many other micro-organisms, are growing in altogether abnormal numbers throughout the alimentary tract in this disease.

Our failure to obtain any decidedly beneficial therapeutic action with anti-diphtheritic serum does not disprove the hypothesis that general paralysis derives its distinctive character from a chronic infection by the diphtheria bacillus.

Behring's serum only acts by destroying the toxins generated by the bacillus; it has no action upon the bacillus itself, the destruction of which is left to the ordinary defensive forces. There is evidence that in the general paralytic these defensive forces are already not only being subjected to an excessive strain, but are threatening to become exhausted. It is easy to understand, therefore, that even if we neutralise a certain amount of toxin by means of an antitoxic serum, the organisms are not destroyed by leucocyte action as they are in cases of acute diphtheria successfully treated thereby, and therefore the patient is not permanently benefited. Moreover it is now well recognised that the treatment of acute diphtheria with anti-diphtheritic serum is only markedly beneficial if undertaken at an early stage of the disease. Buchner (18) has quoted the statistics published by the German Imperial Board of Health, from which it appears that when treatment was begun on the first day the mortality was only 6·6 per cent., whereas when begun on the fifth day the mortality was 23·2 per cent., and when begun after that time, 26·9 per cent. Experimental treatment of infected animals affords similar evidence. The same authority has stated that "if infection has occurred some considerable time previously, so that the disease is already advanced, large doses of antitoxin cannot save the

animal. The disturbances which are already in existence in the various cells of the body can no longer be influenced by the antitoxin." It is also to be taken into account that, at least in the later stages of general paralysis, we are dealing with a mixed toxic infection, in the production of which streptococci, colon bacilli and other organisms have a prominent part. What is probably really needed is a bactericidal serum which may be used at an early stage of the disease, alone, or in conjunction with an antitoxic serum. Wassermann's (19) recent success in obtaining such a bactericidal serum may, we venture to think, prove of great importance in the therapeutics of general paralysis. In conclusion we would say that the hypothesis which seems to be warranted by the results of our investigations is, that general paralysis is the result of a chronic toxic infection from the respiratory and alimentary tracts, permitted by general and local impairment of the defences against bacteria, and dependent upon the excessive development of various bacterial forms, but especially upon the abundant growth of a Klebs-Löffler bacillus of modified virulence, which gives the disease its special paralytic character.

This hypothesis is not proved by the results of our investigations; it is merely suggested and strongly supported by them. Whether it is to be abandoned or retained must depend upon the evidence of the experimental enquiry, for the undertaking of which they have given a clear indication.

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## THE DIAGNOSTIC VALUE OF THE PLANTAR REFLEX.

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THE abnormal type of plantar reflex, described by Babinski in 1896, occurring in lesions of the pyramidal fibres, in brain or cord, has been universally recognised as a sign of the utmost importance in the diagnosis of organic lesions of the brain or cord, involving those fibres either directly or by indirect pressure. So absolute indeed is the acceptance of this "Babinski sign," that it is now practically universally regarded, with the exception of its normal occurrence in young infants, as an infallible sign of organic nervous disease, and, *vice versa*, that a flexor plantar reflex is never found in organic lesions of these fibres, such as in hemiplegia and myelitis. A recent *ex cathedra* expression of this general opinion was given last August at the discussion on Functional Paralysis opened by Dr Buzzard in the Section of Medicine of the British Medical Association meeting at Manchester, in which all the speakers, with the notable exception of Dr Judson Bury, agreed that the Babinski sign was never found apart from organic lesions.

This paper is written in no iconoclastic spirit, but with the object of drawing attention to variations in the type of the extensor plantar reflex, and of placing on record four cases, two of hemiplegia, and two of functional paralysis, which deviated from the accepted rule. In both the cases of hemiplegia a typical flexor plantar reflex was obtained, in each case constant at several examinations during several weeks, while in both cases of functional paralysis a typical Babinski extensor plantar reflex was obtained on both sides, in no way differing in character from

that usually met with in organic hemiplegia or spastic paraplegia. Incidentally I would observe that these exceptions to the general rule illustrate the fact that there is no such thing as absolutism in clinical medicine, and that the Babinski sign, like ankle-clonus and the once so-called infallible signs of meningitis or peritonitis, has its exceptions.

I would here remark that the proper appreciation of the plantar reflex in a doubtful case is often very difficult. In many cases in which its presence would probably be of great service in diagnosis, it is impossible to elicit a satisfactory reflex at all, either flexor or extensor. In two or three cases in which I could obtain no trace of reflex of any kind, the patients declared that their feet were not in the least degree ticklish, and this loss of ticklishness with absent plantar reflexes is met with in some cases of hysterical paralysis as Dr Buzzard long ago noted. In other more sensitive individuals the dorsiflexion of the ankle provoked by stroking the sole of the foot is so brisk and extensive, even on the smallest stimulation, that it is quite impossible to estimate properly the movement of the great toe. In other cases again of early or slight damage to the pyramidal fibres, there may be no plantar reflex obtainable, either flexor or extensor, due, no doubt, to the commencing extensor type counterbalancing the normal flexor reflex, which is, so to speak, on its way to become extensor. This complete absence of reflex, of either type in one foot, associated with a normal flexor reflex in the other, may be of great service in the diagnosis of an early organic hemiplegia, when the weakness is too slight for demonstration, and before the appearance of well-marked exaggeration of the knee-jerk, ankle clonus, or definite extensor plantar reflex. The following case is an illustration of this point. For permission to make use of this case I am indebted to Dr Sidney Phillips, under whose care the boy was in St Mary's Hospital.

C. L., a boy, 12 æt., was admitted to the hospital on 4th Dec. 1901. Four weeks previously he had been seized with sudden nausea and vomiting, which had continued with severe frontal headache, across the forehead. There was convergence of the left eye, due to weakness of the left external rectus, though he was said to have had a squint for years, which had become worse lately. There was very slight right-sided weakness of the arm and leg, only to be detected on careful examination. The knee-

jerks were often unobtainable, no observable difference between the two sides. The left plantar reflex was definitely flexor in type, while the right was doubtful, neither flexor or extensor. No ankle-clonus. He soon became very drowsy and torpid. No fits at any time. On admission the right optic disc appeared congested, and soon developed distinct optic neuritis, which appeared after a week in the left eye also. There were no fits at any time. He gradually became comatose, and died on 6th Jan. 1902. A point of great interest in the history, that was not obtained until after death, was that for the twelve months previous to his admission his moral character had appeared completely and unaccountably changed. Previously an honest lad, attending Sunday school, of steady parents, with no apparent reason he took to lying and stealing.

Post-mortem, a large diffuse glioma was found infiltrating the left frontal lobe, pressing on the pyramidal fibres in the corona radiata.

There was considerable doubt at first as to the localisation of the lesion. Tumour was practically certain from the severe constant headache with progressive torpor and double optic neuritis, though on which side was at first uncertain. The slight right-sided weakness was not obvious to all who examined him, and the clue which practically determined the lesion to be on the left side of the brain was the marked difference between the plantar reflexes, that on the left side being normally flexor, while the right was absent, neither flexor nor extensor movement of the great toe being obtainable. Additional points of interest were the definite commencement of the optic neuritis in the right eye, on the opposite side of the lesion, and the striking perversion of moral character which had existed for twelve months previous to the fatal illness. In the light of the post-mortem it is difficult to resist the conclusion that the slow destruction of the left frontal lobe by the growth, which had probably been progressing for many months before definite symptoms appeared, was closely connected with the perversion of the moral sense.

The best way of examining the plantar reflex is to place the person on his side on a couch, with the knee slightly bent, thus relaxing all muscles. He should lie on the right side when the right plantar is being examined, and similarly on his left side for the left plantar. The dorsum of the foot should be gripped fairly

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tightly, so as to prevent as far as possible dorsiflexion of the ankle from obscuring the movement of the toes. The inside of the sole should then be lightly scraped with a blunt pointed piece of wood, such as the end of a penholder, better slightly roughened, and the strength of the stimulus must be graduated so as to provoke movement of the great toe, but not dorsiflexion of the ankle. It is the movement of the great toe that is of importance, not of the other toes, as their movement seems less constant than that of the hallux, for in some cases of spastic paraplegia, with a typical extension reflex of the great toe, the smaller toes will flex, and in other cases all the toes will give the extensor movement. Sometimes a better result is obtained by stroking across the sole just behind the ball of the foot, instead of along its inner side. In other cases no result may be obtained with a slight stimulation, but on increasing slightly the strength of the stimulus, marked dorsiflexion of the ankle at once occurs, and obscures the true reflex; but this is less likely to occur if lying on the side than if lying on the back when the muscles are less relaxed. The plantar reflex is a much more difficult reflex to test and to appreciate at its true value than is the knee-jerk or the pupil reflex, though they too often want most careful testing. After two years' experience as Medical Registrar at St Mary's Hospital I have found that the average ward clerk's notes are quite worthless on the subject of the plantar reflex, though he may make fair notes on the knee-jerk and the pupil reflex to light.

The rapidity of the extensor movement of the great toe, as well as the amount of the movement, varies in different cases. A rather slow extensor movement is the more typical, and this is the type usually met with in marked cases of spastic paralysis, in which there is little or no voluntary power of movement of the legs remaining. This slowness of the movement I believe to be due to alteration in the muscle tonus, inasmuch as the faradic reactions of the anterior tibial muscles in these cases are always sluggish. In early cases of spastic paraplegia, with little or no rigidity, on the other hand, the extensor movement is brisk, the angle of movement of the great toe varying in extent with the degree of the stimulus employed. The following case is a typical example of this brisk type of extensor plantar reflex :—

K. D., a single woman, æt. 29, sent to me April 14th, 1903, by Mr John Harper of Barnstaple. Since the summer of 1901 she had noticed gradual progressive weakness of both legs, without any pain. There was no numbness or loss of feeling, but she noticed that the legs used to fly about somewhat, making her unsteady. For the last twelve months there had been some loss of control of the bladder. She had been treated by massage for six weeks, but there was no improvement. On examination the gait was ataxic and slightly spastic. Romberg's sign present. There was definite slight loss of power of all movements of the lower extremities, but there was no trace of anæsthesia or analgesia. Eyesight perfect, no diplopia; no contraction of visual fields. Pupils normal; no nystagmus. No weakness or ataxy of arms, and she could write perfectly, thread a needle, and do up all her buttons without the slightest difficulty. No girdle pain. Spine appeared normal. The knee-jerks were brisk, and one or two oscillations of ankle-clonus were obtained on each side. Lying on her side on a couch, with shoes and stockings taken off, there was brisk extensor movement of the great toe on each side on stroking the sole of the foot in the way described above. There was no movement at all of the small toes, but the up and down movements of the great toes were quite rapid, the angle of movement being as much as 30 degrees on increasing the strength of the stimulus.

The diagnosis in this case is clearly an organic paraplegia, not functional paralysis. The progressive weakness of the legs, with spastic ataxic gait and bladder trouble definitely indicate an organic lesion of the cord, which the bilateral extensor plantar reflex confirms. It is more open to question what is the precise nature of the cord lesion, whether it should be labelled "Ataxic Paraplegia" or "Combined Sclerosis," or whether it is not really a paraplegic form of disseminated sclerosis.

It is often said that an extensor plantar reflex is a sign of lateral sclerosis. This is not strictly accurate, for it may make its appearance in an hour or two after the cord has been injured. In a case of fracture dislocation of the spine at the level of the sixth cervical segment which I saw two hours after the injury, there was complete flaccid paralysis and anæsthesia below the second rib, with absent knee-jerks. There was definite bilateral extensor plantar reflex, slight in extent, and somewhat sluggish. In another case of fracture dislocation of the spine in the upper dorsal region in a woman who fell from a second story window,

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there was complete paralysis for many weeks of the lower extremities, with at first absent knee-jerks. For the first week the plantar reflexes were flexor in type, much to my surprise, but then became extensor, and remained of that type. Later the knee-jerks returned, and ankle-clonus and spasticity of the legs appeared.

That a flexor plantar reflex may occasionally be met with in hemiplegia with marked rigidity is illustrated by the following two cases :—

C. W. C., a coachbuilder, æt. 53, had a "stroke" in Sept. 1900, accompanied with loss of consciousness and paralysis down the left side. When seen in Sept. 1902 there was marked post-hemiplegic rigidity. He walked with great difficulty, and had no voluntary power of dorsiflexion of the left ankle or toes. There was regular slow flexion of the great toe on plantar stimulation, sometimes also of the smaller toes. This was found constant at several examinations during two months.

J. N., æt. 42, came to the hospital for epilepsy and paralysis, Maida Vale, Feb. 4th, 1903, with marked right hemiplegia with rigidity due to syphilitic endarteritis. Five years previously he had acquired syphilis, and was treated for two years at a special hospital. In November 1901 began to suffer from constant severe pain at the back of the head, until March 25th, 1902, when his right leg began to drag. He returned home, and as he went up to bed his right arm was getting weak, and he gradually lost consciousness in three-quarters of an hour from the onset of the weakness. He was unconscious for four days, and when he recovered he could not move the leg, and he was partially aphasic for several weeks, using wrong words, which he still does occasionally. The arm has improved more than the leg, and now he can walk slowly, dragging the foot, and there is considerable rigidity of both arm and leg. Can move fingers stiffly. Slight right hemianæsthesia, no hemianopia. The right knee-jerk is much increased, with right ankle-clonus. He cannot dorsiflex the right ankle or toes. There is a definite slow flexor plantar reflex on the right side, which has been constantly present on four occasions since at intervals of a fortnight. There is generally slight dorsiflexion of the ankle when stroking the sole provokes any reflex,

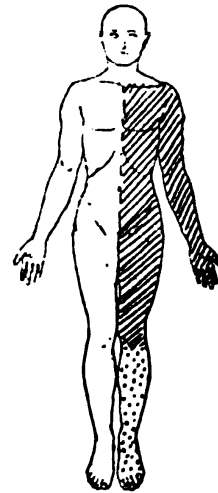
but the movement of the great toe is always flexion. These patients were so obviously the subjects of organic post-hemiplegic rigidity, that the type of flexor plantar reflex present in them was merely a curiosity, and could not for a moment complicate the diagnosis. Far otherwise was it the case in the following two cases, in which, in addition to evidences of functional paralysis, one paraplegic, the other hemiplegic, there was present bilateral typical extensor plantar reflex in each case. This obscured the diagnosis for several days, until the course of the cases cleared up all doubt and proved them to be purely functional.

J. S., a hawker, æt. 25, a small, ill-developed man, was admitted to St Mary's Hospital, October 29th, 1901, under Dr Cheadle, to whom I am indebted for permission to make use of the case. He was admitted for paralysis of both legs and loss of voice. Twelve months previously, when walking in the street, his legs suddenly gave way under him, and he fell down. For eight weeks he had pins and needles sensation in both legs, when the pains suddenly left him, and the legs became numb. Since then sensation has completely returned, but he has been quite unable to use his legs, and he gets about on crutches. Never any bladder trouble or girdle pain. A week before admission he completely lost his voice. On examination in bed the legs seemed strong for all movements, but his attempts to walk were suggestive of "functional" gait, the legs being held stiffly, and dragged along with apparent great effort, not at all suggestive of a true spastic gait. There was no anæsthesia. Knee-jerks brisk and equal, no ankle-clonus. There was bilateral brisk extensor plantar reflex, both great toes being briskly extended, without any dorsiflexion of the ankle, but slight flexion of the small toes. He was completely aphonic, due to functional adductor paralysis, the cords lying wide apart, with very slight movement only on attempts at phonation, though he could adduct them perfectly in the act of coughing. The eye movements were normal, and the visual fields were not contracted. The diagnosis of functional paralysis and aphonia was made, with the reservation that there was probably some organic spinal disease behind it, on account of the typical extensor plantar reflex. He was sent down to me in the electrical department, and I applied strong faradism with a wire brush for about five minutes, suggesting to him at the same time that it would cure him. The effect was immediate, and he was able to walk moderately well upstairs to the ward, carrying his crutches over his shoulder. The aphonia was also cured and did not return. His gait improved day by day until at the end of a week he could walk perfectly. The extensor

plantar reflex persisted unaltered for a few days, but at the end of a week it was found to be of the normal flexor type, and remained so. He was seen again in July 1902, but there was no recurrence of the paralysis or aphonia, and the plantar reflexes were still flexor.

This was the first case I had seen which impressed upon me that an apparently typical bilateral extensor plantar reflex is not necessarily incompatible with the presence of functional paralysis and the absence of organic nervous disease. It might be urged in criticism that my original supposition was the correct one, that the man had functional paralysis complicating some latent organic spinal disease, such as disseminated sclerosis. Against this view I hold that the absence of all signs of organic disease, together with the return of the plantar reflexes to the flexor type, are sufficient proof to the contrary. The only other case I have seen of typical extensor plantar reflex in functional paralysis occurred in an even more striking case of functional hemiplegia.

W. W., a boy of 13, was admitted to St Mary's on June 30th, 1902, under Dr Lees, who has kindly permitted me to make use of the case. On June 23rd he was carrying coals upstairs with his left hand when he felt a sudden numbness and loss of power in the left hand and arm and numbness down the left leg and side. He dropped his burden, supporting himself by the balusters. He walked home, and then involuntary twitching of the left arm, from the shoulder downward, commenced, occurring three or four times every hour. Next day the arm was weaker and the twitching worse, the leg remaining the same. On the 25th the arm was worse and the leg became weak, so that he limped, the numbness on the left side also being worse. The following day he could not walk without support, and he was still weaker. From the 26th to the day of admission he remained the same. No headache or sickness, and he stated positively that previous to the attack he had felt as well as ever since he had had scarlet fever fifteen months



CASE OF W. W. To illustrate anesthesia which is incomplete below the knee.

before, followed for a short time by bilateral otorrhœa. On examination there was weakness of all movements of the left arm and leg, with hemianæsthesia to the mid line involving the left arm and leg and left side of the body below the level of the clavicle and just above the shoulder. There was no anæsthesia of the head or neck, and it was less complete below the knee. Involuntary spasmodic twitchings of the left arm were frequent, especially when under observation. The knee-jerks were equal and normal, but there was fairly well marked left ankle-clonus, and typical bilateral extensor plantar reflex, brisk extensor movement of the great toes, without any dorsiflexion of the ankle. The optic discs, eye movements and visual fields were normal. No pyrexia. Two days after admission he complained of frontal headache and was sick once. He looked distressed, and lay curled up in bed. On the 5th of July the headache had gone, and the anæsthesia disappeared from the left leg and arm, but still present on the left side of the trunk. Still well marked bilateral extensor plantar reflex. On the 7th there was complete flaccid paralysis of the left arm and leg, and the anæsthesia had returned, with complete loss of sense of position of the arm and leg. There was no diaphragmatic breathing, respiration being entirely costal and by the accessory muscles. On the following day the anæsthesia had again disappeared, but the breathing remained as on the 7th. Left ankle-clonus and extensor plantar as before. No optic neuritis. On waking on the morning of the 12th he found he had power to move his left arm and leg. The power gradually increased, and in a few days he was walking perfectly, all the signs of weakness and anæsthesia having disappeared, and he was discharged cured July 22nd. The extensor plantar had now changed to the flexor type, and when seen recently, April 28, 1903, he had remained perfectly well, and there was no sign of weakness or anæsthesia, the plantar reflexes being now brisk flexor in type.

In this case, again, the presence of the extensor plantar reflex strongly suggested an organic lesion, in spite of the limited form of hemianæsthesia, which was clearly functional. The boy's father has always limped badly from old infantile paralysis, so unconscious imitation may possibly have tended to produce the unilateral weakness with dragging of the foot in the boy. The fact of the knee-jerks being equal and normal in spite of the left-

sided weakness and the left ankle-clonus also was in favour of functional paralysis. The left-sided twitching did not resemble the unilateral convulsive movement of cortical irritation, its irregularity and its increase under observation resembling functional tremor. However, the sudden onset of the symptoms and the development of the paralysis and headache strongly suggested an intracranial lesion like tumour or abscess. In both of the cases related the extensor plantar was bilateral, although in the case of the boy the weakness was unilateral. That point is worthy of notice, and might be of service on another occasion in distinguishing a functional hemiplegia from organic disease.

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### **A CASE OF DOUBLE PARALYSIS OF THE LATERAL CONJUGATE DEVIATION OF THE EYES.**

By ALEXANDER BRUCE, M.D., F.R.C.P.E.

CASES of double paralysis of the conjugate deviation are of comparatively infrequent occurrence, and since the one here recorded has been carefully observed clinically almost throughout its whole course, and as an opportunity was afforded of investigating its cause after death, it is thought that the record of the case and of the post-mortem examination may prove of value.

J. F., single, 23, was admitted under my care to the Royal Infirmary, on March 21st, 1902, on account of vertigo and some defect of the movements of the eyes which had lasted for two months.

No definite hereditary tendencies could be traced. Her home surroundings and social relationships were in every respect satisfactory. As regards previous illnesses, she had suffered at the age of 15 from chlorosis, which had tended to recur, and to be associated with somewhat frequent attacks of fainting and vertigo. These, however, had passed off at the beginning of the present year. She had also suffered from three attacks of influenza, the first of which occurred three years ago. In October 1901, she had a slight attack of right-sided pleurisy. Apart from this, patient considered herself in fairly good health.

*History of present illness.*—Patient attributed her illness to an accident which occurred in the middle of January, when she fell

from a ladder and struck her head against the leg of a table. She was stunned and remained unconscious for about twenty minutes. On the following day she felt slightly giddy when walking about, especially on going out into the street. She stated that there was a tendency to sway to the right side, and that this feeling of vertigo was quite different from her sensations in her previous attacks. On consulting Dr Wolston, she was recommended to Dr J. V. Paterson to have her eyesight tested. From the latter I have ascertained, subsequent to the patient's death, that she complained of a giddy, confused feeling on looking at objects, especially when they were at her right side. This feeling was most marked when she was out of doors. She ascribed her troubles to a severe fall on the left side of the head, sustained a week previously. There was no limitation of the ocular movements in any direction. Slight nystagmus was noted on a wide excursion of the eyes being made to either side. The Maddox rod gave a crossed diplopia, increased on looking to the right. Dr Paterson diagnosed a paresis of the left internal rectus, and ordered rest, iodide of potassium, and a blister. This was followed by slight temporary improvement.

Three weeks after the accident, however, severe headaches, with a feeling of nausea, began to trouble the patient, and she complained of difficulty in moving the eyes, especially when she looked at anything to her left side, and she felt some stiffness in the mouth, as though it were pulled to the right, especially in speaking. She also complained of a feeling of formication on the left side of her head, face, and nose, and also of ringing in her ears. Nine weeks after her illness patient consulted Mr George Berry, on whose recommendation she was placed under my care.

*General Appearance.*—Patient is tall, well-developed, and of a healthy complexion. The eyeballs are slightly prominent; the left palpable aperture somewhat more widely open than the right; the left side of the forehead slightly smoother; the mouth slightly drawn to the right, with obliteration of the left nasolabial fold. As regards the facial movements, the left eye cannot be completely shut, and there are the usual indications of a marked degree of paresis of the left facial muscles. The tongue is protruded in the middle line. The pupils are equal. The axes of the eyes are not quite parallel, the left eye being slightly

inverted. When the patient looks at any object straight in front of her, the face is turned slightly to the left. The movements of elevation and depression of the eyes are quite normal. When the attempt is made to look to the left, the left eye does not travel beyond the middle line. The right eye can be made to pass the middle line slightly, so that the two eyes become almost parallel. When the patient tries to look to the right, the right eye just fails to reach the outer canthus, and nystagmoid movements appear as the limit is approached; the left eye moves synchronously with the right one for a short distance, then becomes suddenly arrested in its course, and cannot by the strongest effort of volition be made to reach the inner canthus. Similar nystagmoid jerkings appear when the lateral movement is checked. Convergence is normal if the object is in the middle line, or on the right side; it is less perfect if the object is on the left side. The pupils are dilated. Their reactions are normal both to light and to accommodation. The visual acuity is normal for reading;  $\frac{5}{8}$ ths in both eyes for distance. Colour vision, field of vision, and ophthalmoscopic examination are all normal. Cross diplopia is evident on looking to the right. The cerebral functions, speech, and all the sensory functions, including the muscular sense, the sense of position, and the special senses are normal. Reflex actions; organic, skin and tendon reflexes are all normal. No increase of knee-jerk, no ankle-clonus, no Babinski sign were present. With the exception of the paresis of the left face, and of the movements of the eyes above described, no paralysis whatever was present. Although patient says that she feels a tendency to sway to the right, there is no evidence of this in her gait, which appeared in every respect normal. She can turn quickly without sign of vertigo. When she stands with her eyes shut, she sways slightly. Circulatory and respiratory systems are practically normal. The red blood corpuscles are 3,220,000; the white blood corpuscles 7000; hæmoglobin 70 per cent. The catamenia, however, have been somewhat irregular since they appeared at the age of 16.

From the above account it will be seen that the principal symptoms in the case were:—

- (a) Paresis of the left side of the face;
- (b) Complete paralysis of the conjugate movement of the eyes to the left;

- (c) Slight paresis of the conjugate movement of the eyes to the right; with
- (d) A more marked partial paralysis of the left internal rectus on looking to the right;
- (e) Conservation of all the other ocular movements, including convergence;
- (f) Vertigo, formication of the left side of face, ringing of ears.

The point which presented the greatest difficulty of explanation was the behaviour of the left eye when the patient attempted to look to the right side. That eye, it will be remembered, began to move along with its fellow, but it stopped when about half way towards the inner canthus, and then became the subject of nystagmoid jerkings. This restricted movement was not due to any affection of the internal rectus muscle, or of its nerve or nerve nucleus, for convergence was perfect when the eyes were directed to a near object. The only satisfactory explanation appeared to be that the connection between the right sixth nucleus and the nucleus for the left internal rectus, which is affected by the right posterior longitudinal fasciculus, was partially interfered with. Assuming this to be the case, and that the paresis of the right conjugate deviation was due to a slight implication of the right sixth nucleus, the diagnosis was made of a lesion destroying the left sixth nucleus, infiltrating the left facial nerve in its course round this nucleus, and influencing some part of the left fifth nerve, and also passing across the raphe so as to implicate the right longitudinal fasciculus, and, to a lesser extent, the right sixth nucleus. The lesion was obviously of small size, as it had not in any way involved the pyramidal tract, or any of the other motor cranial nerves. The fact that the lesion was not limited to one-sixth nucleus, and that the paralysis of conjugate deviation was not sudden, tended to negative the idea of a hæmorrhage into the sixth nucleus. It seemed doubtful whether it was in any way directly the result of the accident, and the opinion was expressed that it was probably either of tubercular or gliomatous nature.

Iodide of potassium was prescribed, with absolute rest in bed. By the 10th of April there was a distinct improvement in the symptoms. The vertigo had diminished, the facial



paralysis was less marked, to the extent that the eye could be completely closed, and on frowning there was a distinct, though slight appearance of movement in the corrugator supercilii of the left side. The left eyebrow could also be raised to a slight extent. While there was no change in the movements of the eyes to the left, on looking to the right side the excursion of the right eye towards the outer canthus was greater, although not quite complete, and the range of movement of the left eye inwards was distinctly increased, while the nystagmoid movements were less manifest. On the 30th April, however, vertigo and the feeling of nausea returned, along with slight headache and some depression of spirits, the facial and eye-movements remaining much as on the 10th of April. Subsequently, there was a slight further improvement as regards the vertigo, nausea and headache, up to the 10th of May, after which date ringing sensations returned in both ears, and the headache became worse. On the 16th of May a creeping sensation was complained of along the left side of the head and face. During the rest of the month of May and the first week of June the condition remained much the same as above noticed, with the exception that there appeared to be a distinct diminution of the left facial paresis.

On June 10, a new phase of the illness appeared, which proved to be the beginning of the complication which was soon to cause the patient's death. The record of the case states that patient had an attack of shivering, as if she had got a chill. On June 11, she vomited all afternoon. Her headache became more severe. It was noted that both eyes appeared more prominent, or, at least, that both palpable apertures were more widely open, and that for the last few days the power of conjugate deviation of the eyes to the right seemed to be less than it had been previously.

On the four following days the headache, vomiting and irregular pyrexia continued, and on one or two occasions an attack of shivering occurred. On June 19, the pupils were noted as being unequal, that of the right eye being smaller. The temperature rose to 103°. There was some quiet delirium. The headache became more severe, and there was pain in the back of the neck, aggravated on movement of the head. The neck was kept fixed in consequence. It was noted also that patient appeared to look straight before her, turning her eyes to

neither side, but owing to the distress which any examination caused her, the amount of diminution of movement to the right side could not be accurately tested. It is certain that during the last few days this paralysis of the conjugate deviation had been increasing.

June 25—Increase of headache, and pain in back and front of neck. She wandered in her talk, but when spoken to answered correctly and recognised those about her. Later in the day she became more drowsy. On June 26, this state deepened to complete unconsciousness, and cyanosis developed, the breathing remaining as it had been throughout quiet and natural. She died suddenly at 12.30 P.M.

The symptoms which appeared on June 10 soon indicated a localised meningitic attack, with a deeper involvement of the right sixth nucleus. The occurrence of meningitis suggested that the original lesion was of tubercular origin.

*Autopsy.*—Permission was obtained to examine the brain. There was obviously a gelatinous infiltration of the leptomeninges covering the pons varolii, the medulla oblongata, the corpora quadrigemina, and the roof of the fourth ventricle, which subsequently proved to be tubercular in nature. The thickening of the membranes was most marked in the latter site, but the foramen of Magendie was not completely occluded.

The brain was placed without further preliminary examination in 10% solution of formalin, and after fourteen days transverse sections were made through it. In the upper part of the pons a small tumour was found. It projected into, and almost completely filled the upper part of the fourth ventricle, and occupied only the posterior part of the pons. Its site will be best understood from the two figures (1 and 2), which are made from photographs of the sections. It was ovoid in outline. The antero-posterior diameter measured 1.2 cm.; the transverse 1 cm. It occupied the position of the two abducens nuclei, and the facial nerves, as well as the posterior longitudinal fasciculi, but it did not reach the fillet, so far at least as could be seen macroscopically. The pyramidal tracts were entirely unaffected, and so also were the superficial and deep transverse fibres of the pons. The tumour involved the left side of the pons to a rather greater extent than the right, and extended as far as the nucleus of Deiters, and the sensory nucleus of the fifth nerve, but without,

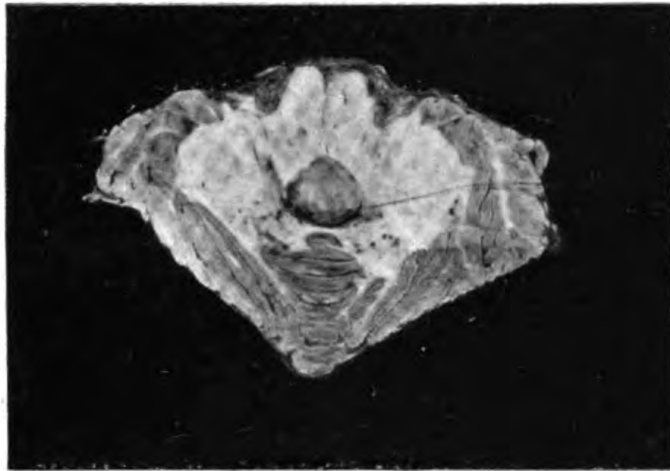


FIG. 1.



FIG. 2.



FIG. 3.

FIGS. 1 and 2.—From photographs of the tumour, to show its relation to the pons and from the ventricle.

FIG. 3.—From a drawing of a Marchi preparation, to show the position of the degenerated fibres in the posterior part of the longitudinal fasciculus, and the manner in which they terminate in the third nuclei.



as subsequent microscopic examination confirmed, destroying these. The tumour was covered posteriorly by ependyma. It was caseous in the centre, with a vascular gelatinous looking margin, and was obviously of tubercular nature.

Thin slices of the isthmus, and of the medulla were placed into Marchi's fluid, and were then cut into microscopic sections in the usual manner. Ascending and descending degenerations were found in the posterior longitudinal fasciculus. The descending degeneration was traced into the antero-lateral tract of the upper part of the cord. It was distinct, but the degenerated fibres were not very numerous. The ascending degeneration was very well seen in both posterior longitudinal fasciculi. As will be seen from figure 3, it is more pronounced in the inner and most dorsal part of the fasciculi. Below the level of the third nuclei no indication of any decussation of the fibres of the two fasciculi could be seen. At the level of the third nuclei the degenerated fibres could be traced into the lower part of the nucleus. Careful examination failed to reveal any evidence of degeneration in either third nerve. A few black granules, it is true, were scattered among the fibres of the roots here and there, but these were always of very small size, and were certainly not more numerous than in any other part of the section. Such appearances are now well known to occur independently of any degeneration. I could not satisfy myself that any degenerated fibres crossed the median plane within the nucleus itself. The sections available all indicated that there was no crossing of the degenerated fibres of the fasciculus, but on the contrary that these (with an exception to be noted immediately) terminated in the nucleus of the same side. It was seen also that it was in the lower and inner part of the nucleus that the degenerated fibres lost themselves; the upper part of the nucleus (comprising the postero-external and internal groups of cells) was free. The upper extremity of the fasciculus was also devoid of degenerated fibres. Opposite the middle of the longitudinal extent of the third nucleus, a small number of degenerated fibres made their way backwards and outwards towards the posterior commissure. Their ultimate destination could not be traced.

One of the points of special interest in the microscopical examination of this case is the light which is thrown on the mechanism by which the movement of conjugate deviation is

effected. The view originally suggested in 1858 by Foville, that the abducens nuclei are the lower centres for the lateral conjugate deviation of the eyes, has been firmly established by a sufficient number of experimental and of combined clinical and pathological observations (1); but the exact path by which each abducens nucleus controls the opposite internal rectus is not yet completely determined. While there is a fairly general consensus of opinion that the fibres which form the first part of this path, after leaving the abducens nucleus, ascend within the posterior longitudinal fasciculus, there is no such agreement as to whether they enter directly into the third nerve and pass by it to the internal rectus, or whether they terminate in the nucleus of the third nerve. Further, it is not settled, in the latter case, whether they end in the third nucleus of the same side, or whether they cross over to that of the opposite side, and if they do pass over to the other side whether they do so at the level of the abducens or of the oculo-motor nucleus or at some intermediate point.

The view that the posterior longitudinal fasciculus contains any ascending fibres has been contested in recent years by such authorities as van Gehuchten (2) and Held (3). They maintain that it is composed entirely of descending fibres, derived according to the former from the "nucleus of the posterior longitudinal fasciculus," and according to Held from the anterior corpus quadrigemina. In face of the degenerations which have been traced upwards after experimental lesions by Thomas (4), E. H. Fraser (5), Risien Russell (6), Mott (7), Gee and Tooth (8), Long (9), and in the above recorded case by the writer, the opinion of van Gehuchten and Held must be regarded as erroneous, and it must be admitted as being beyond doubt that the posterior longitudinal fasciculus does contain ascending fibres. Since these ascending fibres may be degenerated in lesions of the sixth nucleus, it might seem natural to infer that they originate this nucleus. This inference is not warranted, however. Thomas, Risien Russell, E. H. Fraser and others have shown that the nucleus of Deiters sends fibres which pass inwards through the substance of the sixth nucleus and in its immediate proximity—fibres which bend upwards and pass by one or both longitudinal fasciculi to end in the oculo-motor nuclei. After a destructive lesion of Deiters' nucleus the degeneration of these fibres can be traced upwards, through the paths just indicated, as far as the

oculo-motor nuclei. It must be borne in mind, therefore, that any ascending degeneration within the upper part of the posterior longitudinal fasciculus resulting from a destructive lesion of the sixth nucleus must contain fibres which emanate from the nucleus of Deiters as well as those which originate in the sixth nucleus itself. (Indeed a further investigation by means of Nissl's method of the condition of the cells of this nucleus after section of the posterior longitudinal fasciculus between it and the third nucleus is necessary to determine with absolute certainty whether they give rise to any ascending fibres.) Assuming, however, that the sixth nucleus is a source of such fibres, do these enter the third nerve directly, or do they end in the third nucleus? Gee and Tooth (8) traced, by the aid of Marchi's method, a degeneration from the fasciculus directly into the third nerve. On the other hand, however, Wernicke, in a case of complete unilateral paralysis of conjugate deviation, found both nerves quite normal. Long (9), also, in a case of unilateral glioma of the pons, failed to find any degenerated fibres passing from the longitudinal fasciculus into the opposite third nerve, and he points out that the fine granules found in their case by Gee and Tooth among its root fibres do not necessarily bear the interpretation that these authors put upon them, but that they are frequently present even in normal nerves. Thirdly, the case just recorded by the writer is decisive on this point. The degenerated fibres could be traced from the fasciculi into the nuclei, but there evidently ended, without entering the third nerve at all. These three cases make it clear that the connection of the abducens nucleus with the opposite one is an indirect, not a direct one.

With regard to the site of crossing of the fibres to the opposite side, the data of the writer's case are insufficient to enable him to make a definite statement. Duval and Laborde's statement that there is a decussation at the upper part of the pons cannot be supported by the method they employed. A certain proof can be obtained only from the examination by Marchi's method of the ascending degeneration which follows a lesion destroying the sixth nucleus, always, of course, bearing in mind that the ascending fibres from Deiters' nucleus which would be affected by such a lesion cross at or near the plane of the abducens nuclei. There is some experimental evidence (Ferrier and Turner) that the crossing takes place in the upper part of

the pons. In the present case there was no indication of crossing of any fibres from one fasciculus to another; indeed the evidence pointed rather the other way. It is now well known that some of the fibres of the third nerve arise from the nucleus of the opposite side. If it be proved that these are the fibres for the internal rectus, no crossing of the fibres of the fasciculus would be required. The crossing would be infra-nuclear.

However that may be, it seems clear that the crossing takes place considerably above the level of the sixth nucleus, and the exact method of its occurrence, therefore, does not affect the interpretation of the clinical features of the case. The peculiar paresis of the left internal rectus noted by Dr J. V. Paterson as the initial symptom, and as being present only when the eyes were directed to the right side, can now be attributed to a partial lesion of the right longitudinal fasciculus. The subsequent growth of the tumour affected the left side to a greater extent, and the fluctuation of the symptoms can best be explained by the view that the tumour infiltrated the structures that it invaded before it destroyed them, and that the rest which the patient was compelled to take had reduced the congestion of the infiltrating margin and thus diminished pressure.

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## Abstracts

### ANATOMY.

**FURTHER CONTRIBUTIONS TO THE DEVELOPMENTAL (MYELO-  
(185) GENETIC) REGIONS OF THE HUMAN CEREBRAL  
CORTEX.** P. FLECHSIG, *Neurol. Centralbl.*, March 1, 1903,  
p. 202.

In this short paper Flechsig gives the results of a study of six more brains with respect to the date of the appearance of myelin



sheaths in various tracts of fibres proceeding from or to the cortex. This makes a total of 52 on which his conclusions have been based, and the addition of the last six has given him further information, which makes it necessary for him to slightly modify his previous statements.

1. As regards the number of regions into which the cortex may be divided, these were stated by him in 1898 to be 40, but in 1901 he had reduced them to 36. He does not now seek to alter this number, though he admits that subsequent work may yet cause him to do so.

In the first parietal convolution he has hitherto distinguished two regions. He thinks now that there are three. On its outer surface, towards the interparietal sulcus, there is a region which is distinguished by the very early myelination of its fibres, so that it must be considered as one of the primary regions. He gives it now as No. 14. The sheaths are apparent at birth, but do not as a rule become distinctly marked until four weeks later. It is then possible to fix its limits, which form about a tenth part of the first parietal convolution. This region closely resembles in the size and arrangement of the fibres the subangular convolution, and in both the sheaths appear about the same time. He is unable to say yet whether this region contains any part of the fibres of the corona radiata. In newly-born infants the impression is obtained that certain fibres pass from the ascending parietal convolution towards this region. A very evident bundle also appears early and passes radially towards the corona radiata of the posterior part of the gyrus fornicatus, and looks as if it mixed with it. This can be shown to be part of the callosal system, lying between the bundles from each gyrus angularis, which are late in obtaining their sheaths.

The upper and anterior ends of the first occipital convolution were formerly considered as belonging to the sphere of sight. In 1901, however, they were separated by Flechsig and given different numbers. He now finds this conclusion confirmed and in addition differentiates still another bundle, so that the sight region is much more complex than he had at first thought.

2. As regards the order of development of the various regions he considers the following observation of importance. In larger and more fully developed fetuses the motor convolutions show the strongest development of the nerve sheath; but in one 34 cm. long, which he has now had the opportunity of examining, he finds that there is a well marked bundle of fibres with sheaths traceable to the olfactory sphere, while there is no differentiation of the motor tracts. Unless this case is exceptional, the olfactory sphere must henceforth be designated as No. 1, and the central convolutions as No. 2. He desires further investigation of this matter before

changing the enumeration. He thinks it is even possible there may be a regression of some of the olfactory fibres.

3. Flechsig is of the opinion that it is possible to distinguish between motor and sensory bundles of fibres, and that there is a type for each. In the supra and sub-angular convolutions, however, neither type is present. The fibres there exhibit a radial arrangement which, to a certain extent, resembles the motor type, but has important differences. It rather resembles the type found in the callosal fibres.

4. In 1901 the author demonstrated that to every sensory (centripetal) path there corresponds a motor one. He now considers that there is every reason to believe that the projection system of the brain cortex is composed of similar conjugate pairs of bundles.

5. His next point is in connection with the order of development of the brain sulci. He thinks they have a close relation to the order of development of the sheaths in the various tracts which he has discovered. The sulci in the regions which he designates primary appear very early, in those he names terminal they appear last of all. There are apparent contradictions to this supposition, and he desires further investigation, but he regards the relation as of a fundamental nature.

JAS. MIDDLEMASS.

**UPON THE DORSAL LONGITUDINAL BUNDLE, THE RETICULAR FORMATION OF THE PONS AND MEDULLA, THE CENTRAL TEGMENTAL TRACT AND HELWEG'S TRIANGULAR TRACT.** ANDRÉ THOMAS, *Rev. Neurol.*, Jan. 31, 1903, p. 94.

THE author has made an investigation upon a case of hæmorrhage into the pons.

The lesion almost completely destroyed the tegmentum upon one side at the level of the upper limit of the sixth nucleus. It extended mesially to the middle line and outwards as far as the lateral fillet. It extended backwards to destroy the knee of the facial nerve and forwards to involve the mesial fillet and touched upon the most dorsally situated fibres of the pyramidal tracts.

Clinically the case was associated with classic symptoms; paralysis of the opposite limbs; palsy of the face of the same side; loss of conjugate eye movements to the opposite side and conjugate deviation to the same side.

The dorsal longitudinal bundle upon the side of the lesion, was completely degenerated above the level of the lesion and could be traced to the region of the fourth and third nuclei. Below the lesion a few rapidly disappearing fibres alone were degenerated.

There was marked degeneration below the lesion of fibres lying in the reticular formation between the dorsal longitudinal bundle and the mesial end of the middle fillet and this degeneration was traceable into the ventral columns of the cord = ventral longitudinal bundle.

There was marked retrograde degeneration of the fillet on the side of the lesion traceable across the middle line in the fillet crossway to the nuclei of Goll and Burdach.

The central tegmental tract was completely atrophied on the side of and below the lesion and ended in the white coat of the inferior olive of the same side.

Helweg's triangular tract was markedly degenerate upon the side of the lesion. The inferior olive showed marked cell degeneration as is usual when the central tegmental tract is involved.

JAMES COLLIER.

**REMARKS ON THE DORSAL SPINO-CEREBELLAR TRACT.** By (187) C. S. SHERRINGTON and E. E. LASLETT, *Journ. of Physiol.*, vol. xxix., p. 188.

IN the dog, the animal here used, Clarke's column (from the cells of which the fibres of this tract are believed to take origin) extends from the third lumbar to the second thoracic segment inclusive. In order to determine whether there is any definite grouping of the fibres within the tract, the authors in one experiment divided the cord between the thirteenth and twelfth thoracic nerve roots, and on tracing the resulting degeneration forwards, they found that the fibres (which of course belonged to cells situated behind the lesion) formed a thin layer at the surface of the dorsal part of the lateral column and were not scattered irregularly over the whole cross area of the tract. In a second experiment the thirteenth thoracic spinal segment was "exsected," and 270 days later, by which time the fibres arising behind the lesion had disappeared, a partial transverse section was made on the left side, in the anterior part of the tenth thoracic segment, the animal being killed twenty days after the second operation. On making sections at different levels from below upwards the recently degenerated fibres taking origin in the twelfth, eleventh and posterior part of the tenth thoracic segments were found to gradually "slope away from the grey matter . . . and to apply themselves as a thin layer to the deep face of the scar that marks the former site of the fibres of lumbo-sacral origin." In a third experiment the cord was cut completely across at the middle of the second thoracic segment, and 260 days afterwards a transection was made

in the eighth cervical segment, the animal again being killed twenty days after the second operation. In the cervical region, above the second lesion, some freshly degenerated fibres were found to run upwards and outwards away from the grey matter, as in experiment two, until at the level of the third cervical segment they formed a band of degeneration along the deep face of the scar left by the old degeneration. In both experiments two and three the scar and the recent degeneration occupied the same relative position in the restiform body as they did in the spinal cord. The scar resulting from the old degeneration in experiment three extended deeper into the lateral column than the combined areas of the scar and fresh degeneration in experiment two, this being due to the fact that it involved fibres from cells in the eighth to the second thoracic segments inclusive, which were not involved by the lesions in experiment two.

From these three experiments the authors conclude that the fibres of the dorsal cerebellar tract are arranged in definite groups within the tract according to the segmental level of their origin. In other words, the tract is stratified; the fibres from segments farthest backwards lie most superficially, and in proportion as they arise from segments farther and farther forwards they take up a more and more mesial position within the tract, the innermost layer consisting of fibres from the uppermost thoracic and lowest cervical segments. Thus, the further the fibres run the more superficial do they lie. The same general arrangement has been noted previously by the authors in the long descending association fibres of the cord, and by Cohnstamm in Gower's tract.

Further, as the result of transverse section of the cord the large cells of Clarke's column behind the lesion showed chromatolysis and ultimately complete atrophy, but notwithstanding this fact there was no evidence of any degeneration in the fibres of the dorsal cerebellar tract behind the lesion, although these might be expected to show "some participation in the retrogressive changes so evident in the cell column from which they spring." To test the condition of the fibres further, a right lateral transection was made in the tenth thoracic segment 260 days after complete transection of the second thoracic segment. The animal was killed twenty days after the second operation, and by the Marchi method the right dorsal cerebellar tract between the two lesions was found to be extensively degenerated. It would appear, therefore, that atrophy of the cells of Clarke's column resulting from transverse sections of the cord in the lower cervical and upper thoracic segments leads to no obvious degeneration in the main body of the fibres of the direct cerebellar tract, and further, "that after severe atrophy of Clarke's column has set in and become established, transection of the tract in the very region of atrophy of the cell

column still causes full Wallerian degeneration of the fibres headwards of the transection."

Lastly, considering the diminution in size of the degeneration scar of the dorsal cerebellar tract as it is traced forwards in the cervical region, the authors are led to believe that all the fibres of the tract are not destined to reach the cerebellum, and they suggest that many of them are merely "long internuncial fibres ascending from hindward segments to reach spinal segments further forward."

SUTHERLAND SIMPSON.

#### INVESTIGATIONS ON THE COURSE OF THE UPPER AUDITORY

(188) TRACT. A. VAN GEHUCHTEN, *Névraxe*, vol. iv. f. 3, 1902, p. 253.

THIS paper contains the results of a series of experiments, extending over a period of three years, which have led Van Gehuchten to take a view with regard to the composition, course and termination of the central auditory path different to that generally held at the present time. The experiments were performed on rabbits, and the resulting material was prepared by Marchi's method; the usefulness of which method Van Gehuchten praises highly.

In the first group of cases the injury was restricted to the more ventral fibres of the corpus trapezoides, the lesion being produced by tearing out the seventh nerve through the stylomastoid foramen. In this way an injury was inflicted on the surface of the bulb, at the level of emergence of this nerve, with resulting division of a number of the fibres, which, arising in the ventral nucleus of the eighth nerve, sweep downwards through the root fibres of the seventh nerve to enter the corpus trapezoides. Some damage is also inflicted by such a lesion on the fibres of Monakow's and Gower's tracts. On examining serial sections in a case in which such a lesion had been made degenerated fibres were found sweeping into the corpus trapezoides for some distance, vertically, above and below the actual level of the lesion. In the corpus trapezoides these fibres were traced across the middle line to the opposite side; they turned upwards, the more ventral in front of and the more dorsal behind the nucleus of the corpus trapezoides. These two bundles soon united to form a compact semilunar strand (*faisceau arqué*) lying in contact with the outer aspect of the inferior nucleus of the lateral fillet. As this strand was followed upwards it was found to turn outwards and then backwards to enter the lateral fillet, of which its fibres form the more superficial part. In the fillet these fibres were traceable to the base of the posterior corpus quadrigeminum, where they ended in relation to the superior nucleus of the lateral fillet. No degenerated fibres could be traced

to the nucleus of the corpus quadrigeminum itself, nor could any be followed to a higher level in the brain.

These results were confirmed by the examination of a series of sagittal sections from a similar case.

In another series of animals a lesion was directly inflicted on the lateral aspect of the bulb, just ventral to the accessory nucleus. The resulting degenerations were in all respects similar to those just described. No altered fibres were found in the lateral fillet of the same side such as have been described by Ferrier and Turner.

In a third series of experiments the lesion was established by cutting downwards and outwards through the floor of the fourth ventricle so as to divide both dorsal and ventral fibres of the corpus trapezoides. The course of the ventral strands was as already noted. The dorsal fibres were found as a thin strand lying posterior to the olivary nuclei. These fibres were traced across the middle line in the more dorsal part of the corpus trapezoides; they then turned upwards along the posterior aspect of the inferior nucleus of the lateral fillet; ultimately they entered into the formation of the deeper portion of this bundle. They could be traced into the superior nucleus of the lateral fillet, in which, like the ventral fibres, they appeared to terminate.

In a fourth group of cases Van Gehuchten aimed at determining the cells of origin of these ventral and dorsal strands of the corpus trapezoides. He found that a lesion entirely restricted to the accessory nucleus of the eighth nerve produced exactly the degenerations described above; a large ventral strand passing through the corpus trapezoides to end in the superior nucleus of the lateral fillet, and a much finer dorsal strand running a separate course to the same termination. There was no degeneration in the striæ medullares in this group. The homolateral fillet was intact, and no degenerated fibres could be traced above the level of the posterior corpora quadrigemina.

In a further series of experiments Van Gehuchten investigated the relations of the striæ medullares to the central auditory path. Attempts to cut these on the floor of the fourth ventricle were uniformly unsuccessful, but they were found severed in a case in which the seventh nerve had been torn out, and, having ruptured at its genu, had thus accidentally caused their division. From the point of injury degenerated fibres could be followed through the raphe, in which they crossed dorsal to the uppermost fibres of the corpus trapezoides, until they reached a position just posterior to the olivary nuclei. They then turned upwards, forming a well-marked strand, lying a little behind the dorsal tract from the corpus trapezoides, and internal and ventral to the motor nucleus of the fifth nerve. At the level of the upper border of the middle

cerebellar peduncle they swept backwards into the lateral fillet, in which they were found to occupy the deeper zone, being mingled in this part of their course with the fibres of dorsal strand of the corpus trapezoides. Through the lateral fillet these fibres passed to end in the actual nucleus of the posterior corpus quadrigeminum itself; in other words, these fibres of the central auditory path could be traced to a higher level than those previously described. Neither in the ventral nor in this, the dorsal, auditory path, could any connection be traced to the anterior corpora quadregimina or cerebral cortex, such as is described by Held.

In conclusion, Van Gehuchten points out that the central auditory path is a "bulbo-mesencephalic," not a "bulbo-cortical" one. It is double, consisting of a ventral part, arising from the cells of the accessory nucleus, and passing by the corpus trapezoides, and a dorsal portion, arising in the cells of the lateral tubercle, and passing through the auditory striæ. In both cases the connections established are entirely crossed. With regard to the further course of the central auditory path brainwards, Van Gehuchten states that he is at present engaged on a research dealing with the degenerations resulting on destruction of the superior nucleus of the lateral fillet.

This valuable paper is illustrated by numerous diagrams and drawings of the sections described, and contains full references to the works of previous observers.

E. HEWAT FRASER.

#### **THE SPINAL CORD OF THE "PLAGIOSTOMEN" (MYLIOBATIS).**

(189) M. SCHACHERL, *Arb. aus dem Neurolog. Institute in Wien*, H. ix., 1902, S. 405.

In his paper S. gives a detailed descriptive account of the spinal cord of this Australian fish. The spinal cord of fishes show considerable individual variation and that of the Plagiostomata differs from all other fishes.

The cord had a length of 65.5 centimetres, and S. was able to divide it into 120 segments. From these he cut and examined 6000 sections stained by the methods of Weigert-Pal, Van Gieson, etc.

The cord showed a distinct anterior median fissure in its whole extent, while the posterior was well marked only in the middle third of the cord. In the upper part of the cord also, situated dorsolaterally on transverse section, is a third furrow or fissure, which he says has occasionally been found in man.

Microscopically the cells in the anterior and posterior horns of grey matter show no definite grouping. They are triangular in shape and have fine granules.

The spinal cord of this fish differs from that of other described fishes in that the grey matter is broken up in a peculiar manner. In sections of the first segment of the cord appear two bundles of white matter of considerable size (paramedianes längsbündels) lying laterally one on each side of the grey commissure. A little lower down in the cord these became more strongly marked and divide the commissure into a ventral and a dorsal part. The central canal lies in the ventral grey commissure close to the bottom of the anterior median fissure. Lower still in the cord they became so marked as to divide the grey matter into five separate parts. In the caudal region these individual portions of grey matter became approximated. The blood vessels of the cord are very tortuous.

EDWIN MATTHEW.

**DESCENDING DEGENERATIONS SECONDARY TO A SOFTENING OF THE CEREBRAL PEDUNCLE.** CESTAN, *Rev. Neurol.*, fev. 28, 1903, p. 195.

A CASE of Weber's syndrome due to softening of the left cerebral peduncle, the result of thrombosis in the posterior cerebral artery, which lived only one month after the onset, furnished suitable material for anatomical investigation with Marchi's method.

The lesion, which completely destroyed the crus, nucleus ruber, fillet and left oculomotor nucleus, was accurately limited by the middle line, and did not extend into the pons. Only the upper two segments of the spinal cord were examined.

Besides that of the pyramidal tracts, some degeneration was found in the opposite anterior cerebellar brachium, in both dorsal longitudinal bundles, but chiefly in the homolateral, and in the interolivary layer of the same side. The latter fibres cross the raphé in the medulla, and end in the opposite nucleus gracilis.

Other degenerated fibres which descend through the tegmentum of the pons form a compact bundle in the bulb, dorsal and lateral to the inferior olive (central tegmental tract).

Degenerated bundles were also visible in both anterolateral columns of the spinal cord, larger on the side of the lesion. In the first cervical segment each lay along the ventral margin opposite the anterior horns, but with their dorsal ends overlapped by the direct cerebellar tracts. These bundles could be traced from the site of the lesion, first ventral to the anterior cerebellar brachium, then in the tegmentum of the pons, and on the lateral margins of the medulla dorsal to the olives. As they have been found degenerated in other cases referred to, in which the pyramidal tracts were intact, the authors regard them as descending fibres from the mid-brain probably from the nucleus ruber.



The position of this tract in the section of the cord figured certainly does not correspond to that of the tractus rubrospinalis but rather to the ventro-lateral pyramidal fibres.

GORDON HOLMES.

### PHYSIOLOGY.

#### **THE INHIBITORY FIBRES OF THE HEART BELONG TO THE (191) VAGUS NERVE AND NOT TO THE SPINAL ACCESSORY.**

A. VAN GEHUCHTEN, *Névrose*, vol. iv., f. 3, 1903, p. 303.

THE old view that the inhibitory fibres of the heart arose from the medulla oblongata by the spinal accessory nerve and passed thence by its internal branch to join the vagus was founded on experiments by Waller and afterwards supported by numerous observers. Van Gehuchten reviews the history of these experiments minutely. Bischoff experimenting on dogs came to the conclusion that the spinal accessory was purely a motor, and the vagus purely a sensory nerve. Claude Bernard tore out the spinal accessory nerve as it left the skull and found subsequent paralysis of the muscles of the larynx, but no alteration in the movements of the digestive, circulatory or respiratory organs. Waller concluded that the motor fibres of the heart and stomach contained in the vagus as well as the greater part of the motor fibres of the larynx were derived from the spinal accessory. Van Gehuchten states that the method of operation employed by these observers is a very difficult one in the animals they experimented on, but is comparatively simple in the rabbit. He used a large number of rabbits and divided the fibres of origin both in the spinal canal and in the cranial cavity. He confirms the view which ascribes to the spinal fibres of the spinal accessory the motor supply of the sterno-cleido-mastoid and trapezius muscles; section of these fibres in the spinal canal produces no degeneration in the internal branch of the spinal accessory. Section of the bulbar rootlets, however, gives rise to degenerative changes in the internal branch which joins the vagus, and the degenerated fibres can be traced down the cervical portion of the vagus into the inferior laryngeal nerve, and go exclusively to supply the external thyro-arytenoid muscle. There are no degenerated fibres in the vagus below the inferior laryngeal nerve, so that none of the bulbar fibres of the spinal accessory go to the heart. After section of the bulbar fibres of the spinal accessory stimulation of the vagus in the neck on the same side two to six weeks after the operation gives the usual inhibition of the heart.

The author also employed the method of Bernard, tearing

away the rootlets of the spinal accessory, and obtained contradictory results in different animals; this he ascribed to the method which often injures the neighbouring rootlets of the vagus. Finally he made use of the method of stimulation of the individual rootlets of the spinal accessory. He also discusses the grouping of these rootlets with reference to the glossopharyngeal, vagus and spinal accessory nerves, and criticises the divisions made by Grossmann, Rethi and Kreidl. At the end of the paper are a series of diagrams showing the grouping of the rootlets in several animals.

The author concludes: (1) that the inhibitory fibres of the heart do not come from the bulbar fibres of the spinal accessory, but belong to the vagus itself.

(2) All the bulbar fibres of the spinal accessory go to constitute the inferior laryngeal nerve.

(3) The subdivision of the rootlets of the bulb into three groups made by Grossmann is arbitrary and unnecessary.

(4) The origin of the eleventh pair of nerves is as Willis said exclusively spinal.

PERCY T. HERRING.

#### ON MORPHOLOGICAL CHANGES IN EXHAUSTED GANGLION

(192) **CELLS.** GORDON HOLMES, *Ztschr. f. allg. Physiol.*, Bd. 2, S. 502.

THE material on which these observations were made consisted of the central nervous systems of about twenty of the frogs used by Verworn\* in his investigations on the vital processes of nerve cells.

To produce severe degrees of fatigue and exhaustion large doses of strychnine were administered, and when the resulting tetanic spasms had ceased and the reflex irritability was lost, owing to accumulation of paralysing metabolic products in the cells, consequent on cardiac paralysis (fatigue), an artificial circulation of oxygen free salt solution was started. The spasms and reflex irritability at once return, but soon again disappear, only to reappear when an oxygenated artificial circulation is substituted. But finally, after perhaps twelve hours, the spasms again cease, and do not return even on replacing the salt solution by blood. This is the final stage of exhaustion due to complete combustion of the nutritive store of the cells, as Verworn has conclusively proved by other ingenious experiments.

The nervous systems of these so-treated animals were examined by several modifications of Nissl's method. The most pronounced changes were found in the ventral cornual cells of the spinal cord. In the early stages only some rarification of the tigroid round the nucleus was visible. Later the nucleus enlarged, the tigroid began to disappear from all parts of the cells, latest from the periphery and dendrites, and in the stage of complete exhaustion a few

\* *Archiv. f. Anat. u. Physiol. Physiol. Abth.* 1900.

isolated and irregular clumps could be seen only in the dendrites. In the earlier stages the cell protoplasm takes a deep and finely granular stain, but later it is palely and homogeneously tinted. The enlarged nucleus becomes excentric, in the later stages forms a hernia of the cell wall, and finally ruptures. This state represents complete and irreparable degeneration.

These experiments differ materially from all others in which strychnine has been used to produce cell changes, as the artificial circulation enabled a much greater amount to be administered over a much longer period.

These changes might be attributed to three different causes, (1) to direct action of the strychnine; (2) to an osmotic action of the salt solution; or (3) to the overwork and deficient nutrition to which the cells were subjected.

Any direct action which strychnine might have was easily differentiated from the effect of the overwork due to the associated spasms, by suppressing the latter by immersing frogs to whom many times the ordinary lethal dose had been administered in water at 0° C. As no change could be found in the nerve cells of animals so treated a direct action of strychnine can be positively excluded. Control experiments in which strychnine was not used showed no cell changes, so an osmotic or other action of the salt solution does not come into question, and the conclusion must be drawn that overwork when associated with deficient nutrition can cause severe morphological changes, in and finally complete degeneration of nerve cells subjected to their influence.

Such a result is possible under much less severe conditions in mammals whose cell life is much more intense, and may be active in the pathogenesis of certain degenerative diseases.

AUTHOR'S ABSTRACT.

## **PATHOLOGY.**

**THE PATHOLOGY AND MORBID HISTOLOGY OF JUVENILE (193) GENERAL PARALYSIS.** GEORGE A. WATSON, *Archives of Neurology*, vol. ii., 1903, p. 621.

THIS paper is founded upon the family, personal and clinical histories, and upon a study of the morbid anatomy and histology of twelve cases.

The chief factors concerned in the genesis of the disease are concluded to be heredity and syphilis, both probably being essential. The syphilitic factor may have to be taken into account twice; first as a devitalising influence on the parents, and secondly as an actual infection of the offspring.

The essential feature of the disease from the side of its patho-

logical histology is a slow primary progressive decay of the neurone owing to its inherited defective durability, and some cases show practically only this change. To this however is added, in the majority of cases, a more acute or subacute destructive process, dependent chiefly upon vascular changes. Proliferation in and about the vessel walls and glial development are not on the whole such prominent features as in most adult cases. In the cases which show merely chronic degeneration of the neurone the above alterations are comparatively slight. It is concluded that the more chronic changes found in the vessel walls are largely the result of an old standing reaction to the damage produced by syphilis, and that much of the more recent vascular and interstitial proliferation is a further reaction, following or concomitant with the neurone degeneration, and is also of the nature of an effort at repair. The signs of this latter reaction are always most intense in situations where the neurone destruction is most acute.

The disease in its later stages is a widespread one but the neurones are not affected indiscriminately. Apart from certain phenomena of syphilitic origin or dependent upon gross vascular disturbances a certain "selection" of the degenerative process is evident, which corresponds closely with the order of development of the neurones and with their relative functional value. Of the cerebral cortical cell layers the pyramidal is always the most affected by the chronic degenerative changes, especially the smaller and medium-sized cells of this layer. Of the cortical nerve fibres the super-radial and tangential systems are more profoundly atrophied than are the deeper horizontal and radial systems. The "coarse" fibres of the former systems seem to be earlier affected than the finer ones. The cerebral cortical areas, so far as examined, are placed in the following order as regards relative degree of involvement:—

1. The association areas of Flechsig, the anterior practically always most.
2. Broca's convolutions, with perhaps the posterior portion of the second frontal.
3. The central convolutions, the posterior usually more than the anterior.
4. The occipital region, which, however, is always more or less affected.

AUTHOR'S ABSTRACT.

### CLINICAL NEUROLOGY.

**TOXIC DEGENERATION OF THE LOWER NEURONES SIMULATING PERIPHERAL NEURITIS.** STANLEY BARNES, *Brain*, Winter 1902, p. 479.

UNDER this title are described seven cases of a rather unusual type. In all the cases the most marked feature was an atrophic paralysis,

symmetrical in distribution and severely affecting the intrinsic muscles of the hands. The sensory signs were much slighter than is usually seen in alcoholic neuritis, and contracture only occurred to a slight degree in one of the seven cases. Five of the cases gave a history of some febrile condition preceding the onset of the disease, viz., rheumatism (in two cases), diphtheria, mumps and some pyrexial disease of unknown origin in one case each; one was possibly alcoholic, and the seventh of unknown origin. The onset of the paralytic condition usually took place in the third or fourth weeks after the fever; the course was sub-acute or chronic, but (with one exception) when once improvement began it was continued to a slow recovery.

The one case, after two early relapses, died in hospital of a third attack, after the memoir was written, but the results of the histological examination are given in an appendix, and bear out the views which had been expressed by the author in discussing the pathology of the condition. The conclusions are:—

(1) There is a clinical type which is usually the sequel of acute specific fevers, which resembles the paralysis seen in multiple neuritis, but which is associated with great atrophy of the hand muscles. It usually begins about the second or third week after the febrile condition, and involves the muscles from the periphery to the trunk to a varying extent. It may progress for a few days only, or for several months. Sensory signs are present, but slight in degree. Although the condition somewhat resembles progressive muscular atrophy, the prognosis and etiology of the two conditions are probably widely different from one another.

(2) After a certain stage, when once definite improvement has begun, relapses are not common, and there is a constant tendency to improvement. Even years after the subsidence of the acute condition considerable improvement may still take place, the small muscles of the hands being the last to recover. Contractures are rarely developed.

(3) Probably the condition is one of toxic degeneration of the lower neurones, the motor neurones being more particularly affected.

Special stress is laid upon the similarity of these cases in their onset to Landry's paralysis, the more acute ones giving a history almost identical with that of a case of the latter disease; the circumstances of death in case 3 were almost identical in character and duration with those of a patient dying in the hospital at the same time of Landry's paralysis. The cases were distinguished from multiple neuritis clinically by the severe atrophy of the hand muscles, the slight tendency to contractures and the slight sensory signs; and histologically, by the absence of any signs, either in the spinal cord or nerves, of a true inflammation.

AUTHOR'S ABSTRACT.

**SOME OBSERVATIONS ON PRIMARY DEGENERATION OF THE  
(195) MOTOR TRACT.** F. W. MOTT and A. F. TREDGOLD, *Brain*,  
1902, p. 407.

THE authors record, with admirable minuteness, the pathological appearances found by them in the nervous system in four cases :— two of amyotrophic lateral sclerosis, one of progressive muscular atrophy, and one of chronic rheumatoid arthritis with marked muscular atrophy. In the first case of amyotrophic lateral sclerosis, they found, in addition to atrophy of the anterior horn cells of the affected levels of the cord and bulb, degeneration of the large cells of the motor cortex and of the efferent tracts throughout the capsule, pons, medulla and cord. In the second case of amyotrophic lateral sclerosis, besides the atrophy of anterior horns and of bulbar nuclei, there was sclerosis throughout the pyramidal tracts, direct and crossed, also recent degeneration of projection-fibres in the motor area of the brain, but no alteration in the cortical motor cells. In addition there was sclerosis in the postero-median columns of the cord and in the ascending antero-lateral tracts. In the case of progressive muscular atrophy, the usual degeneration was present in the anterior horn cells and anterior nerve-roots of the cervical region. In addition there was slight sclerosis in the lateral columns, confined to the lumbo-sacral region, no change being found in the pyramidal tracts above that level. There was sclerosis in the antero-lateral regions of the cord, confined to the cervical enlargement and therefore attributed by the authors not to a tract degeneration of the ascending antero-lateral paths but to atrophy of out-going root-fibres in the affected region. Slight sclerosis was also found in the posterior roots and posterior columns throughout the cord.

In the case of chronic rheumatoid arthritis with secondary muscular atrophy in a woman of 53, the only pathological change in the nerve elements consisted in chronic atrophy with excessive pigmentation of the anterior cornual cells of the cervical and lumbar enlargement, all the groups, especially the mesial-anterior, being affected. There was no tract-degeneration but a general slight interstitial overgrowth throughout the cord. Slight chronic changes were present in the posterior ganglion cells; there was some atrophy of peripheral nerve fibres and atrophy and fatty degeneration of the affected muscles.

All four cases also showed universal arterio-capillary fibrosis.

The authors consider these cases as examples of primary degeneration of the motor tract, not secondary to a localised lesion nor part of a general disease.

As to the nature of the morbid process, they point out that either the upper segment alone, the lower segment alone, or both

upper and lower segments simultaneously, may undergo primary degeneration, the change being one of slow decay of nerve-cells, nerve-fibres and muscles, and quite unlike the appearances met with in acute processes. The changes of decay they attribute to "exhaustion of inherited vitality" of the affected neuron. The abundance of intra-cellular pigment in such cases they regard as an indication of pre-senile decay. They combat the view which explains the changes as due to toxic agencies. As to the arterio-capillary fibrosis, they consider that it is not the primary cause of the disease, though it may act as a contributing factor. As to why in some cases the arm fibres, in other the leg fibres, should be mainly or preponderatingly attacked, they regard the occupation of the patient as a determining factor by causing over-use of one particular function (this, by the way, is Edinger's well-known theory); whilst the selection of the tract itself, whether efferent or afferent, must be due, they think, to hereditary disposition.

The changes which occur in such gradual decay of the neuron do not occur simultaneously throughout its extent, but tend to begin in the most distant part of the fibre and ascend gradually towards the cell. This is why in amyotrophic lateral sclerosis degenerative changes of the pyramidal tract are always best marked in the lower part of the cord and diminish as we approach the cortex. Such degeneration may involve either the upper or the lower segment of the motor tract separately or both together.

Finally, the involvement of other neuron systems than the efferent ones in such cases is considered by the authors as a secondary affection of the association fibres and of the afferent systems. Hence the great disappearance of short association fibres from the net-work of the anterior horns and the sclerosis occasionally present in the anterior commissure of the cord. The atrophy of the posterior columns is attributed by the authors to a "disuse-atrophy" of afferent fibres from the muscles. If the pathological changes were confined to the posterior roots corresponding to the muscles affected, such an explanation might possibly suffice, but it seems inadequate to explain the degeneration of posterior roots in *all* regions of the cord, as described by the authors themselves in their case of progressive muscular atrophy of the limbs.

PURVES STEWART.

**A CASE OF AMYOTROPHIC LATERAL SOLEROSIS BEGINNING  
(196) IN THE MEDULLA.** F. FRANCESCHI, *Riv. di Patol. nerv. e  
ment.*, f. 10, 1902, p. 433.

THE disease occurred in a man aged 56, previously quite healthy and of healthy parents. The first symptoms noticed were: Difficulty in pronunciation and in respiration, followed

by weakness in the right hand, then the left, and finally the legs. To these were superadded disturbances of deglutition. On admission to the hospital his condition shortly was as follows: Muscles of lower limbs and trunk rather well preserved, distinct atrophy over the scapular girdle, of the upper arms, and especially of the forearms. The patient could close both eyes perfectly together, but on leaving the left open the closure of the right was imperfect. Movements of the eyeballs were perfect. The teeth were shown symmetrically, but he could not inflate the cheeks properly nor could he whistle. The lateral movements of the jaw were very limited and the uvula prolapsed. The defects in speech and deglutition were quite evident. In the hands movements were weak on account of the atrophy of the interossei and thenar and hypothenar eminences. Six months later the paralysis has advanced considerably. The patient can't close his eyes properly, especially the right, the palate is paralysed with pharyngeal reflex abolished, speech much worse, especially the labial explosives and dentals. In the arms supination is incomplete while abduction and adduction of the hand are impossible. The bicipital, radial and ulnar reflexes are intact. With advance of the disease the affection of the upper extremity and of the face became more marked while the lower limbs began to show evident involvement, and towards the end the legs were rigid and extended with the feet in the position of equino-varus, and the toes over-extended, especially the great one. The plantar and patellar reflexes were exaggerated. The Babinski sign was present, no ankle-clonus, movements of legs slow but not diminished and sensibility unimpaired.

Pathological anatomy: In the ascending frontal convolution the giant cells were reduced in number and in volume, with central chromatolysis. The large pyramidal cells were atrophied. Marchi's method showed black globules in the direction of the radial fibres in the frontal and ascending convolutions. Some neuroglia hypertrophy was present. The other areas of the cortex showed nothing unusual.

In the cerebral peduncles and pons the pyramidal paths were normal. In the motor root of the fifth nerve there was considerable destruction of elements and also in the facial nucleus. The intercerebral tract of the facial was atrophied. In the medulla at the inferior limit of the olive the pyramids were narrowed, and the sclerosis of the left most evident. The dorsal nucleus of the vagus and glossopharyngeal and the nucleus ambiguus were much injured. The hypoglossal nucleus showed the most change of all the bulbar nuclei.

In the spinal chord the cervical portion was most injured. The cells were diminished in number especially in the anterior



horns. Some of the affected cells were in the reactive stage. By Marchi's method degeneration, shown by a few scattered droplets, was present in the pyramidal tracts, and to an even less extent in the direct cerebellar and antero-lateral columns. Weigert's method demonstrated well the sclerosis in the pyramidal tracts, and the atrophy of the anterior roots, anterior commissure, and of the fine intercrossing fibres in the anterior horns. The neuroglia was hypertrophied in the sclerosed areas. In the dorsal cord the cells were very much reduced in numbers, and the sclerosis in the pyramidal tracts and the loss of fibres in the anterior horns and in the anterior commissure were very noticeable.

In the lumbar cord and in the sacral the above-mentioned changes were present but to a much less extent. Distinct atrophy was present in the hypoglossal, median and circumflex nerves. In the peroneal nerve there was extremely little change.

At the end of his paper the author emphasises the bulbar origin of the disease with subsequent spread to the upper limbs and then invasion of the muscles of the back and lower limbs. Particular attention is to be paid to the fact that some cells were found in the reactive stage, due to injury of the axis-cylinder.

The sclerosis is due to a primary affection of the nerve fibres starting in the pyramidal paths at the upper part of the medulla and spreading downwards. After destruction of the nerve sheath the axis-cylinder becomes affected and then the cells react. In this way the affection of the cortical nerve cells is accounted for.

DAVID ORR.

**CYTOLOGICAL EXAMINATION OF THE CEREBRO-SPINAL**  
 (197) **FLUID IN TABES.** ARMAND-DELILLE et JEAN CAMUS.  
*Rev. Neurolog.*, Feb. 1903, p. 199.

THE authors present in this paper the results of an investigation which they carried out, with a view to finding out whether leucocytes were present in the cerebro-spinal fluid in cases of tabes. They systematically examined the fluid obtained from 13 cases of tabes of durations varying from 4 to 42 years. The fluid was obtained by lumbar puncture and was then centrifuged, being afterwards mounted on two slides, dried and fixed in alcohol. One of the preparations was stained with thionine, the other with hæmatoxyline-eosine. Leucocytes were found in some cases of 18 to 19 years' duration but were not found in cases of from 4 to 8 years' duration. The clinical symptoms bore no relation to the presence or absence of leucocytes. Speaking only of those cases which they had examined they state that in their opinion such an investigation as they carried out could be of no diagnostic or prognostic value.

T. GRAINGER STEWART.

**COMPRESSION OF THE SPINAL CORD BY FRACTURE OF THE  
(198) SPINE, SPASTIC PARAPLEGIA, LAMINECTOMY, RECOVERY.** F. RAYMOND et J. A. SICARD, *Rev. Neurolog.*, Feb. 1903, p. 193.

IN this paper there is an interesting case of spastic paraplegia due to fracture of the spine, which was completely cured by operation nine months after the injury.

The patient had fallen some height and injured his back about the region of the 12th dorsal spine, and, although never unconscious, he instantly became completely paraplegic with retention of urine. The condition improved slowly but the patient was unable to walk without assistance on both sides. He was admitted to the Salpêtrière six months after the accident.

At that time his condition was as follows: His general health was good, the lower extremities showed general wasting and considerable spasticity much more marked in the right limb than in the left. There was some contracture which could easily be overcome by passive movement. The abdominal muscles on the right side were slightly contracted. There was no localised weakness. The tendon reflexes were increased especially on the right side and there was ankle-clonus and extensor plantar response on both sides. There was some pain at the level of the right groin and lower abdomen. There was no objective sensory change in the right leg but on the left there was definite loss of all forms of sensation below the groin. He had some trouble in micturition but the sphincter ani was not affected. His gait was spastic and he could only walk a short way with crutches. There was no change in the electrical reactions.

He was operated on three months later, the laminae of the 11th and 12th dorsal vertebrae were found to be fractured. The right side and the broken part of the 12th were displaced and pressing on the cord.

The condition was relieved and the patient recovered from the operation well. Within fifteen hours of the operation all the symptoms had disappeared and the reflexes were normal.

In conclusion the authors point out (1) that the condition which had persisted for seven months was not due to changes in the cord secondary to the compression, and that to have diagnosed such would have been wrong; (2) that the spastic condition does not depend exclusively or necessarily on degeneration of the pyramidal tracts and that such independence may be seen both of cases due to cerebral lesion as well as in cases of compression in the cord.

T. GRAINGER STEWART.

**DISEASE OF THE SUB-OCCIPITAL VERTEBRA, WITH DISLO-  
(199) CATION BACKWARDS OF THE SKULL; BILATERAL  
ATROPHIC PARALYSIS OF THE TONGUE.** DECROLY, *Journ.  
de Neurol.*, Feb. 5, 1903.

THE patient was a man thirty-three years old, who, when raising his head rather quickly, suddenly felt as if he had been struck a smart blow on the top of the head; this was followed by an impression of cold and of tingling in the same region. Two months later the pain shifted to the back of the neck, more especially on the right side, and the condition remained without further change for a year. Then a slow paralysis developed of the left arm and leg, later of the right—only face, tongue, and jaw movements were unimpaired. At the same time there was loss of sensation in all the paralysed areas. At the end of the next six months this paralysis had slowly disappeared, whereas the tongue began to atrophy and became immobile. Eventually, however, the paralysis in the limbs returned again, together with difficulty in speaking and swallowing. The face and neck became wasted. All the deep reflexes were exaggerated. The tongue was now quite atrophied, and the seat of incessant fibrillary twitchings; it could not be protruded beyond the lips. There was anæsthesia in the skin areas supplied by the occipitals, great auricular and cervical plexus; there was no impairment, however, on the face or tongue. On opening the mouth one could see that the pharynx was remarkably narrowed.

The author goes into the pathology of *malum vertebrale sub-occipitale*, and discusses each symptom from the pathological standpoint. He divides his case into three stages: the first being one of inflammation and pachymeningitis, leading to compression; the second, softening of the vertebræ, luxation of the head, and involvement of the hypoglossal nerves—the disappearance of the paralytic symptoms being possibly due to momentary cessation of the inflammatory process; the third, extension of the disintegration, leading to aggravation of the symptoms, the paralysis being no longer due to tubercular infiltration and compression from œdema, but to the actual displacement of the vertebræ.

S. A. K. WILSON.

**HÆMORRHAGE INTO THE RIGHT ANTERIOR CORPUS  
(200) QUADRIGEMINUM.** BOUCHAUD, *Arch. génér. de méd.*, mars  
31, 1903, p. 782.

AFTER commenting on the rarity of lesions confined to the corpora quadrigemina the author describes the case of a man, aged 61, who, apparently healthy, had a sudden apoplectic shock, followed by practically complete paralysis of the right ocular musculature,

at the same time there was disturbance of equilibrium and a tendency to fall to the paralysed side.

At the autopsy six weeks later, the only discoverable pathological condition was a minute capillary hæmorrhage—about the size of a pin-head—in the centre of the right anterior corpus quadrigeminum.

According to Raymond, the symptoms following a lesion in the corpus quadrigemina are four-fold in character ; impairment of sight, of the pupillary reactions, of the ocular muscles and of equilibrium.

As far as the first is concerned, it is more than doubtful if the corpora quadrigemina can be considered as visual centres ; recent researches seem to show that the optic fibres pass to the cortical visual centre by the external geniculate body and the pulvinar ; the patient in question had apparently no affection of sight at all.

The effect on the pupil is usually a unilateral or bilateral mydriasis, with impaired reaction to light and accommodation ; but the author cannot say that his case showed much change in this direction.

The paralysis of the right ocular musculature is much more difficult to explain. The nucleus of the third pair on the right side was not involved, and the hæmorrhage itself was much too small to affect the nucleus by pressure, yet the paralysis was almost complete, involving also muscles supplied by the fourth and sixth. The author inclines to the view of a subnuclear paralysis, following Nothnagel and Von Monakow, according to whom the chief characteristics of such an ophthalmoplegia are the asymmetrical nature of the impairment, some muscles being involved more than others ; a predilection for the superior and inferior recti, ptosis, and absence of associated motor disorders of the other eye.

The staggering gait and tendency to fall to one side are characteristic of a lesion in the cerebellar peduncle, especially the superior. According to Bruns, in a lesion in the cerebellum, ataxia precedes ophthalmoplegia, whereas the reverse is the case if the corpora quadrigemina are involved.

S. A. K. WILSON.

**CASE OF ARRESTED DEVELOPMENT OF THE CEREBELLUM  
(201) AND ITS PEDUNCLES ; WITH SPINA BIFIDA AND OTHER  
DEVELOPMENTAL PECULIARITIES IN THE CORD.** By  
W. B. WARRINGTON and KEITH MONSARRAT, *Brain*, Winter,  
1902, p. 444.

THE patient whose brain and cord are described in the paper was a female child aged six weeks. She was brought to the Liverpool Infirmary for children on August 10, 1901. The mother had one other child living. After marriage she had two miscarriages ; then the elder of the living children was born ; then followed

another miscarriage; and then this child. When brought to the Infirmary she had marked hydrocephalus. In the lumbar region was a scar of a healed spina bifida; she also had right talipes varus, and exhibited no powers of movements in either lower limb. She died from intercurrent gastro-enteritis on August 29th. The naked eye appearances of the brain and cord were as follows:—The cord is an attenuated structure throughout; in the lumbar region it passes into a mass of cicatrix at the level of the spina bifida; in the dorsal region it is divided into equal halves, between which an exostosis projects. The spinal roots and nerves appear normal; around the cord and between it and the dura is a quantity of loose friable tissue. In the medulla there are no prominences corresponding to the anterior pyramid and olive, and the medulla passes insensibly into the pons. The cerebellum is extremely rudimentary, being only represented by a few leaflets. The mesencephalon is represented by a single large structure, no trace of a division into anterior and posterior corpora quadrigemina being seen. The cerebrum exhibits a condition of marked hydrocephalus. On histological examination the following peculiarities were made evident:—

1. *In the brain.*—Arrested development of the cerebellum, and of its efferent and afferent fibres, and of their nuclei of origin. Absence of restiform bodies, the olives, accessory olives, external arcuate fibres and nuclei arcuati. Arrested developments of the middle peduncles and transverse fibres of the pons, pontal nuclei, and vestibulo-cerebellar tracts, of the superior peduncles, grey nuclei of the cerebellum, red nuclei of the tegmentum.

2. *In the cord.*—Marked enlargement and irregular outline of the central canal; normal development of grey matter with well-formed ganglion cells; absence of all medullated fibres below the upper dorsal region; above this, medullated fibres in the postero-external tract and anterior ground substance chiefly. In the lower dorsal region the central parts of the cord are disintegrated and show various irregular cavities; the fibro-cellular tissue in which the cord is embedded is a highly cellular connective tissue containing large vascular channels. The case exhibits, therefore, a condition of arrested development of the whole of the central nervous system from the mesencephalon downwards. The association of this with deformities of the surrounding parts is of importance; that is to say, the spina bifida, the exostosis described, and the transformation of the membranous coverings. The limitation of the medullated fibres has an important bearing on the developmental history of these structures, and the anatomy of the central canal also appears to illustrate points of interest in the formation and changes of this channel. With regard to the condition of the brain, the arrested growth of the cerebellum is the key to most of

the peculiarities in the hind brain. An examination of the literature of cases of cerebellar atrophy shows that from the point of view of their pathogenesis they may be considered as falling into the following classes:—

1. Cerebellum almost completely absent on one or both sides, condition primarily due to arrests of development.

2. Cerebellum congenitally small, but of normal histological structure.

This group includes the cases of Nonne, Miura, and Fraser, and which exhibited during life the symptom group of Marie's hereditary cerebellar ataxia.

3. Comprises the majority of cases. There is an atrophy with a general sclerotic condition. The cortex appears to be primarily affected either in whole or part, the disappearance of white matter corresponding to the extent of cortical defect. There are often indications of chronic meningitis and vascular degeneration. The symptoms are those of epilepsy with mental enfeeblement, and in a number of cases date from the occurrence of some acute infective disease. The vascular changes are usually secondary. Probably an acute disease acts as an exciting agent in developing a previous existing morbid condition, and leading to retrograde changes in the nervous tissue.

4. Primary vascular disease attended by chronic interstitial inflammatory changes, chiefly affecting the white matter, a cirrhosis cerebelli—comparable to cirrhosis of the kidney. This is a rare condition.

5. A primary atrophy affecting the cerebellar-cortex, the nuclei of the pons and in the inferior olives, accompanied by marked atrophy of the middle peduncle and partial atrophy of the restiform body. This also is a rare condition. Three cases have been described by Thomas and Déjerine, and the atrophy of the cells is considered to be comparable to the Duchenne-Aran atrophy of the ventral cornua.

An appendix contains an enumeration of the cases falling in these several categories.

AUTHOR'S ABSTRACT.

**AN AUTOPSY UPON AN ACROMEGALIC GIANT.** P. E. (202) LAUNOIS and PIERRE ROY, *Rev. Neurol.*, Jan. 31, 1903, p. 92.

THE clinical history of this case has been reported in the *Nouvelle Iconographie la Salpêtrière*, July-August 1900.

The subject, who was 2.12 metres tall (83.5 inches), had developed the characteristic symptoms of acromegaly since the age of 21 years, and from that time had suffered with persistent glycosuria. Subsequently he developed signs of tuberculosis at the apex of the left lung.

A few days before death he became mentally apathetic and he died in an attack of general convulsions.

At the autopsy was found a pituitary tumour of soft consistency and of a greyish colour. This tumour consisted of two lobes connected by a narrow pedicle. One lobe occupied a much enlarged sella turcica; the narrow process occupied the infundibulum and the other much larger lobe filled the anterior part of the right lateral ventricle. The histological characters of the tumour showed that it was a primary epithelioma of the glandular portion of the hypophysis. In the spinal cord a remarkable diminution in the ventral horn cells and many small calcareous plates in the pia-arachnoid were found.

Of great interest was the remarkable condition of splanchnomegaly present. The thyroid body was enormously hypertrophied and its structure resembled that of a fibrous goitre. The weights of other organs were as follows: Liver 4650 grammes, spleen 370 grammes, kidneys 325 and 390 grammes, pancreas 250 grammes. These organs presented no peculiarities of anatomical structure. The brain, on the other hand, weighed only 1350 grammes.

It is remarkable that defects of vision and symptoms referable to the presence of a large intracranial tumour were absent in this case. Enlargement of the thyroid body has been frequently reported in cases of acromegaly (13 cases out of 26 Hinsdale.) Splanchnomegaly has been reported in giants by Dana, Buday and Janeso, and by Dalleniaque and in connection with acromegaly by Linsmayer, Bourneville and Regnault and by Chauffard and Ravant.

JAMES COLLIER.

**A CASE OF SENSORY APHASIA FROM A LESION OF THE**  
(203) **RIGHT TEMPORO-PARIETAL LOBE.** M. A. JOFFROY, *Rev.*  
*Neurol.*, Jan. 31, 1903, p. 112.

A RIGHT-HANDED man, aged 77 years, was suddenly seized with mental agitation after which he presented almost complete word deafness and word blindness. He could recognise some letters and could appreciate the meaning of certain words. Paraphasia, paraphasia, and sometimes jargon utterances were present. He could write his name with difficulty but could not copy nor write from dictation. Death occurred from cerebral hæmorrhage three months later. Examination of the brain showed that the left hemisphere was normal. The right hemisphere presented a patch of softening involving part of the posterior third of the first and of the second temporal gyri. Around this patch shrinking of the

convolutions and adherence of the membranes was present, extending over the posterior two-thirds of the first and second temporal gyri and involving the whole angular gyrus.

M. Joffroy is of opinion that this man was congenitally left-handed but that he had become trained to be right-handed, and he offers this explanation for the situation of the speech centres upon the right side of the brain. No ancestral left-handedness is mentioned in the history.

JAMES COLLIER.

**MIRROR WRITING.** ROBERT D. RUDOLF, *Canadian Practitioner and (204) Review*, Feb. 1903.

DR RUDOLF records the case of a lady, aged 29, highly intellectual and an accomplished pianist and linguist. She was markedly left-handed, sewing with her left hand and holding her table-knife in that hand in spite of the fact that ever since childhood she had been most strictly enjoined to use the right. She could not remember when she began to write in mirror fashion, but thinks that she did so the first time she attempted to write at all. At first she always did it with the left hand; with unusual difficulty she learned to write with her right hand in the ordinary manner. At present she can write with either hand in either manner, but left-hand mirror writing is the most easy and natural for her. She can read mirror writing quite easily but not so quickly as the ordinary form; this, Dr Rudolf suggests, is probably to be explained by want of practice.

As to the explanation of ordinary mirror writing, the author states that all the cases on record seem to be cases of writing done with the left hand.

"In a naturally left-handed person, the impression in the right brain is good, and, although such an individual has always been forced to write with his right hand, when anything happens to prevent this, then he has the right brain and left hand to fall back upon, and the result is left-handed (or natural) mirror-writing. This writing is not a diseased condition, but the (to the patient) artificial form of writing being rendered impossible by the disease, as it would be by tying his right hand behind the back, he reverts to his natural type of chirography." This, as the author states, is also the view taken by Professor F. J. Allen (*Brain*, 1896, p. 385) who is himself a mirror writer.

Dr Rudolf refers to Dr Ireland's interesting experiment in which a teacher made all the children in his class write with the left hand. Five wrote backwards, they were all found to be left-handed, and they were the only left-handed children in the class of sixty.

EDWIN BRAMWELL.



**OPHTHALMOPLÉGIA AND ARTERIOSCLEROSIS.** PECHIN et (205) ROLLIN, *Rev. Neurol.*, mars 15, 1903, p. 256.

It is here pointed out that ocular paralysis may be produced by compression of the nerve trunks by diseased and dilated vessels.

The observation on which this view is based was made in a case of tabes with complete external ophthalmoplegia and optic atrophy on the left, and on the right ptosis and paralysis of the superior rectus.

General and severe arteriosclerosis was found in the necroscopy.

The left carotid which was the more severely affected, was enormously dilated in its passage through the cavernous sinus, and there compressed the ocular nerves which run in the outer wall of the sinus.

The left ophthalmic artery was also distended, and compressed the optic nerve.

The same lesions were present, but less pronounced on the right side.

The basilar artery was also enlarged, and may have compressed the oculomotor nerves in the interpeduncular space. No microscopical examination of the intra-medullary portion of the nerve-trunks, or of their nuclei is mentioned. GORDON HOLMES.

**UNILATERAL DILATATION OF THE PUPIL DUE TO A SPIKE (206) OF HORDEUM MURINUM IN THE EXTERNAL EAR MEATUS.** J. LABRAZIS, *Rev. Neurolog.*, Feb. 1903.

THE patient, a man aged 47, was sleeping on the grass when he woke suddenly, feeling something in the right ear which caused buzzing, pain and tenderness, and proved to be a piece of spike of *Hordeum murinum* 2 in. long, lying against the drum.

Before removal of the foreign body it was noticed that the right pupil was much larger than the left, contracting much less also than the left to light, accommodation, convergence or skin stimuli. Two hours after removal the dilatation disappeared and both pupils became of equal size and reacted normally. The only other signs present were slight trembling of the closed lids, and of the fingers when the hands were extended, besides slight evidence of right apical tuberculosis. No evidence of tabes, general paralysis, cerebral tumour or syphilis.

The author explains the unilateral dilatation as being due to a reflex action carried from the external ear by the branches of the auriculo-temporal and also the auricular branch of the vagus which supplies the tympanum.

He cites a similar case by Bamdelier who believes the cervical

sympathetic branches innervating the vessels of the external ear to be the fibres which transmit the stimulus to the iris-dilating fibres of the sympathetic.

In support of his explanation that the case is one of spasmodic dilatation the author cites cases of unilateral dilatation due to middle ear disease, labyrinthine disease and nasal ulceration, and mentions the fact of the case with which the pupil may be dilated by stimulation of the face, neck or forehead of the same side.

Besides pupil dilatation many other phenomena may be produced by foreign bodies in the ear, and the author cites several cases of reflex coughing, epileptiform convulsions, heart palpitation, etc., so produced.

T. GRAINGER STEWART.

**A STUDY OF SENSATIONS IN MOTOR PARALYSIS OF  
(207) CEREBRAL ORIGIN, BASED UPON THIRTY-FIVE  
CASES. ALFRED GORDON, *Journ. Nerv and Ment. Dis.*,  
March 1903, p. 144.**

THIS paper records the results of careful testing of sensation in thirty-five cases of hemiplegia due to organic cerebral disease; only good witnesses for anæsthesia are included, and any cases with hysterical stigmata were excluded.

He finds that astereognosis was complete in 22 cases and partial in 7; pain-sense was lost in 5 and diminished in 25; heat-sense was lost in 8 and diminished in 20; cold-sense was lost in 3 and absent in 17; and touch was lost in 4 and diminished in 19 cases. It was also evident that the anæsthesias became less frequent and less pronounced in the hemiplegias of long standing.

In discussing these results the author strongly supports the doctrine of a sensori-motor Rolandic cortex, and concludes that probably hemi-sensory disturbances always accompany a motor paralysis of cerebral origin. It is evident that the author has obtained his results after very careful testing, so that it is a little surprising that he has not noted any cases in which pain sensation, though much dulled on the arm and forearm, is yet acute on the palm of the hand; but the author has done useful service in drawing attention to the milder degrees of anæsthesia which are so common in hemiplegia, and which are so often overlooked.

STANLEY BARNES.

**SYMPTOMATIC CATALEPSY AND CHEYNE-STOKES RES-  
(208) PIRATION. A. BAUER, *Rev. Neurol.*, mars 15, 1903, p. 249.**

As *Catalepsie symptomatique*, Brissand has for years described a syndrome whose dominant feature is a tendency to maintain the

limbs, etc., in certain positions, as in hysterical catalepsy. He regards cortical insufficiency as the pathological basis of these characteristic symptoms, which may be present in diverse conditions.

Bauer carefully records four cases, two of hemiplegia with chronic nephritis, a case of chronic uræmia, and one of cancer of the œsophagus, in which Cheyne-Stokes respiration was associated with this symptom. The two phenomena may either coexist, alternate or succeed one another. The cataleptic attitudes may be maintained during several respiratory cycles, but almost invariably some relaxation of the active muscles is present during the stage of apnoea. Sometimes the apnoea overtakes an action and checks its performance, the limb affected remaining motionless in any stage of its movement till the commencement of the next respiratory phase.

The deeper the cerebral inhibition within certain limits, the more complete is the muscle relaxation during the apnoëic stage, and consequently the shorter the duration of the cataleptoid attitude.

Stern and Pic have pointed out other rhythmical symptoms—psychomotor, pupillary, circulatory, reflex, etc.—occasionally associated with Cheyne-Stokes respiration, and to be added to these is, in Bauer's opinion, *Catalepsie symptomatique*, which may appear and disappear, or alternate with it, or existing independently of it, may be found associated with the same psychical state, the same general indifference, mental torpor and passive activity.

The ætiological factors of the two conditions seem to be identical; profound cerebral inhibition with cortical insufficiency.

GORDON HOLMES.

## PSYCHIATRY.

**AMENTIA (IDIOCY AND IMBECILITY).** A. F. TREDGOLD, *Archives* (209) of *Neurology*, vol. ii. (pp. 328-424, with 4 plates and numerous tables).

THE writer of this article had access to over 1500 cases of every grade and variety of amentia, and the progress of many of these was carefully noted for a period of nearly two years. He also obtained a complete family history of 150 of these patients, and made a microscopical examination of the nervous system of 12 cases dying during the course of the enquiry. The article is divided into three sections—1, Etiological; 2, Clinical; 3, Pathological.

SECTION I.—ETIOLOGY.—The factors elicited have been grouped into two main classes, viz. (1) *Morbid Hereditary Influences*; (2) *Environment*. Those in the former group are far and away the

most important in causing the various grades of amentia, since it was found that no less than 90 per cent. of the cases could be attributed to their operation. The chief of these hereditary factors were insanity, or other abnormality of the nervous system (82·5 per cent.), alcoholism (46·5 per cent.) and tuberculosis (34 per cent.), and it is worthy of note that in no less than 62 per cent. of those cases with ancestral nervous abnormality, either alcoholism or consumption, or both, were also present. Syphilis, in the absence of antecedent nervous symptoms, was rarely a cause of amentia, and consanguinity, *per se*, appeared to have no influence.

Injurious factors in the environment of the patient, although frequently contributing to the result where morbid heredity was present, were in themselves only responsible for 10 per cent. of the cases; they are considered in three stages—before, during, and after birth. Before birth, the only influence which appeared to have any effect was a general toxæmia of the mother (alcoholism, syphilis, etc.), which was responsible for 1·3 per cent. of cases. The mental state of the mother seemed to have practically no influence, since although maternal impressions, worry, and fright were frequently alleged, the writer found no single case where such occurred in the absence of a pronounced morbid heredity, and by tracing children actually born while the mother was insane, he found that their subsequent mental and physical condition was invariably dependent upon the presence or absence of a pronounced hereditary predisposition, and had no relation to a temporary attack of insanity in the mother. The chief cause acting during birth was protracted delivery, producing asphyxia neonatorum, which was responsible for 2 per cent. Those acting after birth produced 6·7 per cent., and were either idiopathic epilepsy, or some traumatic or post-febrile condition, which in the majority of the cases only gave rise to amentia through the medium of epileptic convulsions.

The figures obtained differ in some respects very considerably from those of previous authors; especially is this so in the extent to which hereditary influences were noticed. This is attributed to the fact that instead of being a compilation from probably incomplete case-books, they are the result of a personal enquiry of the patients' friends, and in most cases particulars were obtained of every member of the family for three generations.

From a general consideration of the figures obtained, the author concludes that amentia is therefore the result of accidental causes in not more than 10 per cent., and that in the remaining 90 per cent. it is no freak and no variation, but the final outcome of definite injurious influences acting upon successive generations. It is considered that the presence of a general toxic condition like alcoholism or tuberculosis tends, in the first instance, to diminish the inherent

vitality of the germinal plasm, giving rise in the offspring to a condition of nervous instability, manifested as hysteria, migraine, etc. By unsuitable marriage the condition becomes accentuated in a subsequent generation, and results in epilepsy or actual insanity, whilst by a further repetition of the process this instability and tendency to degeneration of the nervous system gives rise to an actual imperfection of neuronie development—*amentia*.

SECTION II., which deals with the *classification and varieties* of this condition, divides all the cases into the two groups of (1) *primary amentia*, or those due to inherent defects in the germinal plasm resulting in *imperfect* mental development; and (2) *secondary amentia*, due to the *arrest* of development by extraneous causes. Attention is also drawn to the fact that in nearly half the cases epileptic convulsions occur, and that insanity is very common in the higher types. A table of classification is given, showing the various relations which exist between the etiological factors, pathological conditions, and clinical aspects of the many varieties of *amentia*.

SECTION III.—The results obtained from a microscopical examination of the central nervous systems of 12 of these cases are in complete accord with the etiological deductions and the table of classification, since in cases of primary *amentia* the essential condition present is a numerical deficiency, an irregular arrangement, and an imperfect development of certain of the cortical cells and their processes, and, generally speaking, the degree of *amentia* is in direct proportion to the extent of these microscopical changes, although other pathological conditions are frequently superadded.

In the secondary form, on the other hand, the changes are principally those of degeneration, involving the same cell layers as are imperfectly developed in the primary form, and the author has found that the same neuronie systems are also those chiefly and earliest affected in the dementia following insanity.

AUTHOR'S ABSTRACT.

## TREATMENT.

### THE TREATMENT OF EPILEPSY BY PSYCHICAL METHODS.

(210) H. CAMPBELL THOMSON, *Lancet*, April 18, 1903, p. 1092.

AFTER insisting upon the necessity for basing any methods of psychical treatment upon physiological principles, the author briefly reviews evidence to show that there are certain possible psychical conditions which can tend to arrest, to diminish, or to increase the frequency of fits.

First, some of the methods by which fits are known to be arrested apart from the influence of drugs are considered, such as

the classical instance of tightening a cord round the arm or leg in connection with an aura of the limb, or by the stimulation of other sensory nerves as by smelling pungent salts or by inhalation of amyl nitrite. In many cases the patient finds that he is able to control his fits by an effort of will, and moreover fits seldom occur when the attention is being closely held by any special occupation or other mental effort. On the other hand, mental worry of any kind is very apt to increase the number of fits.

Next the influence of operations upon epileptics in whom nothing abnormal was found or in whom the abnormality had no relation to the epilepsy is considered, and the writings of Dr J. William White are quoted from the *Philadelphia Med. Journ.*, 1901, to show that the most diverse operations appear to have a curative effect in a certain percentage of cases after all doubtful results are excluded—a result which appears to be due to some psychical influence. The views of Dr James Jackson Putnam are also referred to in respect to the treatment of Jacksonian epilepsy, and after a careful consideration of the question that writer states that in his opinion “the arrest of epilepsy through surgical operation is an affair primarily of inhibition, and next of the establishment of a new habit, made possible by this temporary arrest of the morbid outbreaks.”

The treatment of epilepsy by hypnotism is mentioned, and here the author refers especially to the work of Boris Sidis upon Psycho-Pathological Researches in Mental Association in which attempts are made by hypnotism to obtain from the patient all the details which happened during the fit, such memories being, of course, lost during the waking condition. Boris Sidis bases this form of treatment on the idea that in many instances the fundamental lesion is a dissociation of groups of neurons whereby they become separated from the conscious mind although they still exist in the subconscious mind, and an interesting account of a case of epilepsy is given in which this treatment was practised.

Having thus shown that epilepsy can be influenced to a considerable extent by psychical conditions, the author proceeds to consider the best way in which these conditions may be turned to practical advantage, and the first method he advises is based as far as possible upon the first group of causes which tend to arrest the fits. The indications on which to base the treatment according to these lines are to inhibit the attacks either by a voluntary action of the will or by raising the nervous activity of some neighbouring centre. Attempts to inhibit the attacks voluntarily are of great value and should be strongly encouraged. Patients who have well-marked warnings often describe how on some occasions they pull themselves together the moment they feel queer and in this way apparently abort the attack, but they seldom try to cultivate the

habit to any extent or to elaborate it in any way. If the importance of the effort is pointed out to them they take a keen interest in it and make use of it systematically with much greater success than before. This voluntary effort may be further strengthened by methods based on the facts that tightening a cord round the arm will sometimes arrest a fit and that fits do not commonly occur when the attention is occupied. It is probable, the author considers, that in the cord method the inhibition in many cases starts from the brain itself rather than from the periphery, in which case it should be found useful in cases other than those in which the aura begins in the limbs, and particulars of an instance are given in support of this suggestion.

The next method was suggested by a perusal of Boris Sidis' work. As already explained the object of this worker was to bring back to the conscious memory the facts that occurred during unconsciousness, by which means, acting on the theory of "dissociation," he hoped to re-associate the neurons. To accomplish this he employed hypnotism, which for various reasons the author has not personally investigated, but has endeavoured to carry out the principle in a modified manner by encouraging voluntary efforts of memory on the part of the patient during that period of the fit which precedes total loss of consciousness. To carry this out effectually the patient must be carefully instructed to remember every possible detail at the commencement of each fit. It is interesting to see how the power of memory increases and extends further into the fit with practice, and it certainly seems to be a beneficial process. As will be gathered, the fits which are most likely to be benefited by psychical treatment are those in which there is a definite aura. Those who endeavour to carry out the methods certainly appear to be benefited and to be able to abort attacks which they would otherwise have had. Particulars of a case which has been specially studied are then given.

While acknowledging that the methods of inhibition suggested are somewhat crude, the author thinks this mode of treatment has possibilities of a future before it, and that if the exact nature of the warnings is carefully studied many fits may be checked which otherwise would certainly occur.

AUTHOR'S ABSTRACT.

**HYDROTHERAPY IN THE PSYCHOSES.** W. ALTER, *Centralb. f. (211) Nervenheilk. u. Psychiat.*, March 1903, S. 157.

THIS paper gives the results of a year's further experience in the application of hydrotherapy to the treatment of mental disorders, the conclusion arrived at being entirely—indeed almost excessively—favourable to this therapeutic measure, which, it is alleged, "has

in a comparatively short time rendered possible the complete abandonment of isolation, and a substantial reduction in the amount of drugs administered" in the author's department of the provincial asylum at Leubus.

The material consisted of 75 patients belonging to the educated classes, of whom 59, suffering from various forms of psychosis, were submitted to hydrotherapeutic treatment. This consisted of prolonged baths of one to six hours' duration; of continuous baths in which the patients were kept immersed for a period varying from twelve hours to as much as twenty-three days (the last in one instance only); or of wet-packing during one and a half to twelve hours.

The first method was found most useful in the case of wakeful and not very excited patients, in the acute exacerbations of chronic delusional cases, and in the emotional variations of hebephrenics and katatonics.

The continued baths (*i.e.* those of over twelve hours' duration) have been found "an absolutely sovereign remedy" in all states of severe excitement (whether melancholic or maniacal), in acute confusion, and in hysterical exaltation, though the time of application may have to be prolonged. Patients of dirty and careless habits are often improved to a surprising degree, as in the case of two general paralytics specially mentioned, where the dirty and aggressive patients became quiet and clean under this treatment, and slept well. Rapid healing of a severe bed-sore in one case, and even recovery from phthisis in another, are said to have taken place. The bodily weight increased under the treatment.

Wet-packing had much the same effect, but in less marked degree. It produced good nights, but its effects were otherwise transitory, and it was found of most use in quiet sleepless cases and half-quiet general paralytics, and especially with hysterical patients, while it proved better than baths in states of depression without great motor restlessness. A frequent mode of use was to leave the patients in the pack for the night, a plan which the patients themselves often liked. The best temperature for the water is 33° to 34° C., and there should never be profuse diaphoresis. The only contra-indications are certain severe hallucinatory and agitated melancholias, but in these the continuous bath is excellent.

In suitable melancholic cases, the author has found it advantageous to combine increasing doses of opium with systematic wet-packing, and states that in this way the action of the drug is accelerated and increased; and in alternating insanity a regularly intermittent treatment by baths and rest in bed has favourably modified the course of the attacks.



On the financial side, the cost of a bath installation must be weighed against the saving effected in material (owing to diminished destructiveness) and in drugs, and no increase in the asylum personnel has been found necessary. The amount of water required is about 150 to 160 litres per head per day.

As regards the mode of action of hydrotherapeutic measures practically nothing is known, the only useful theory being that of reduction of irritability due to continuous warmth. In an effort, however, to obtain some data the author has made the following observations as to the effect of baths and wet-packing on both healthy and morbid subjects:—

The pulse is almost always slowed, and the pulse-curve shows a flattening of the apex, due to diminished cardiac energy. Blood-pressure falls owing to the same cause with dilatation of the arteries, and the temperature also sinks (even that of the skin showing no rise for many hours).

On the side of the nervous system, electric irritability (both to the galvanic and faradic current) was at first increased, but fell again after some hours, often below the normal level. Tactile sensibility, on the other hand, at first diminished somewhat, but ultimately became greatly augmented. Muscular power, as tested by the dynamometer, was much lessened. In the psychical area simple reactions were facilitated, but the higher associations became impaired, and there was a marked reduction in the power of psychical resistance, the susceptibility to hypnotic and suggestive influences being strikingly increased.

W. R. DAWSON.

#### HOW TO PREVENT THE SPREAD OF PELLAGRA IN EGYPT.

(212) F. M. SANDWITH, *Lancet*, March 14, 1903, p. 723.

PELLAGRA occurs chiefly among the poorer peasants of Lower Egypt who eat diseased maize. Where maize is not the staple diet pellagra is not prevalent. Sandwith examined working men and of these, varying percentages, from 30 to 62, in different villages were pellagrous. All were supposed to be healthy and could do a fair day's work. These are high figures, and if typical of the State of Lower Egypt, require immediate attention. The ignorance of the disease is great. The local authorities are lax in the inspection of maize exposed for sale, and the poorest peasants sow tainted grain, gather it unripe and store in damp places without removing the cob sheath, and the worst maize, too bad for market, is eaten at home. Bad hygiene is also a factor. The Government has a heavy task before it, as the peasants are quite illiterate. The importation of maize from pellagra-infected countries should be controlled.

C. H. G. GOSTWYCK.

## Proceedings of Societies

### **PATHOLOGY AND BACTERIOLOGY OF LANDRY'S PARALYSIS.**

At a meeting of the Neurological Society of the United Kingdom held at 11 Chandos Street, W., on April 2nd, Dr E. Farquhar Buzzard read a paper on the "Pathology and Bacteriology of Landry's Paralysis," and showed microscopical specimens illustrating the same.\*

The first part of the paper comprised a short summary of all the most recent investigations which have been made on this disease and tended to show that from a clinical and pathological point of view the term "Landry's paralysis," as at present used, denoted a symptom-complex rather than a distinct morbid entity. Bacteriology has hitherto failed to throw much light on the nature of the condition, but from a consideration of those cases which have yielded positive bacteriological findings, and from a study of the results obtained by various investigators with experimental inoculations of microbic toxins, Dr Buzzard came to the following conclusions:—

- (a) Even by modern histological methods a few cases of Landry's paralysis present no demonstrable lesions.
- (b) In the large majority of cases the lesions are such as can be produced in the central and peripheral nervous system by the action of microbic toxins apart from the microbes themselves. In these cases it is the rule to find that bacteriological investigations have given negative results.
- (c) In a few cases the lesions are those of a disseminated or diffuse myelitis or meningo-myelitis of varying degrees of intensity, and in some of these, pathogenic organisms have been demonstrated in the meninges, spinal cord and cerebro-spinal fluid, and occasionally in the blood and other organs as well.

The second part of the paper contained the results of an investigation on a fatal case of the disease which was under the care of Sir William Gowers at the National Hospital for the Paralysed and Epileptic, and which ran its course in eighteen days.

The pathological findings consisted of (1) intense engorgement of the vessels of the spinal cord and meninges, (2) slight chromatolytic changes in the ganglion cells of the cord, (3) diffuse Marchi degeneration in the white columns of the cord and in the peripheral nerves and muscles.

\* This paper will appear in full in the spring number of *Brain*.

No bacteria were found in the central or peripheral nervous system nor in the lepto-meninges or cerebro-spinal fluid, but a micrococcus was found in considerable abundance in the outer vascular layer of the spinal theca. What appeared to be the same organism was isolated in pure culture from the heart blood of the patient, but was slow and scanty in its growth. Inoculated subdurally into a rabbit it produced at the end of seven or eight days an ascending flaccid palsy, and post-mortem the same organism was discovered in the spinal dura mater of the animal. The coccus was grown in pure culture from the rabbit's blood and the histological changes in the spinal cord were similar to those found in the human patient.

This coccus was generally seen in pairs or groups of four, each half of the diplococcus being hemispherical in shape and presenting a flattened surface to its fellow. In the primary cultures it stained moderately well with the aniline dyes but did not retain Gram's stain; in later subcultures it retained the colour when treated by Gram's method. The organism was further characterised by very marked pleomorphism and by the fact that it grew only on blood agar and in broth, and even then very slowly. On the former medium it produced greyish-white colonies of rather varying size.

Dr Buzzard concluded by suggesting that the examination of the dura mater might possibly prove to be fruitful, not only in other cases of Landry's paralysis, but also in various diseases of the nervous system which are certainly or probably of microbic origin.

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## Review

**LA PSYCHOLOGIE CRIMINELLE.** Par Prof. PAUL KOVALEVSKY, M.D. Paris: Vigot Frères, 1903.

ACADEMIC discussion has done little towards settling the long-vexed question of the relative importance of education on the one hand, and of innate disposition on the other, in the conduct of human life. The use of such scientific methods as the careful collection of facts and the deduction of inferences from them is of comparatively recent application to the study of crime and criminals. Criminal psychology is one of the youngest aspirants to the dignity of recognition as a science, but already the work of such men as Despine, Thomson, Nicolson, Virgilio, and especially Lombroso and his followers, has made it clear not only that crime as a social phenomenon must be studied in connection with the criminal, but that those who are branded as criminals by the law are far from

forming a homogeneous assemblage. On the contrary, while there are some who seem to come into the world with hereditary tendencies which unfit them for any but a life of crime or which even drive them irresistibly to it, there are others who find themselves in the ranks of crime through causes, educational and other, which may be classed as environmental. To study the criminal with the object of arriving at some satisfactory classification, to discover the causes which lie at the root of the social disorder of which crime is the leading symptom, and to apply suitable methods of prevention and cure, are the problems which the criminal psychologist has to keep before him.

Professor Kovalevsky, in the work before us, adopts the classification of Ferri which recognises four classes of criminal,—the born criminal, the criminal by profession, the insane criminal, and the criminal who has been led astray by accidental causes. It is to the first of these almost exclusively that the work is devoted, but space is found for a discussion of certain mental states which the author regards as closely related to that of the born criminal, namely those characteristic of moral insanity, of hysteria, and of epilepsy.

The writer describes at considerable length the leading features of the character of the born criminal. Among these he notes especially childishness; love of amusements and especially spectacular displays; love of animals and at the same time cruelty to animals, to men, and even to their own relations; a tendency to form societies among themselves; callousness to pain; religiosity; moral insensibility; mental deficiency. After a general discussion of these and other features the author turns to the detailed consideration of special subjects under such headings as: the child assassin; the murderer; the thief; vagabondage; the female criminal; the prostitute. These subjects are treated in an interesting way and the author's handling of controversial questions is marked by a judicial cautiousness and temperance of statement. On the whole it must be admitted that he makes out a good case for the existence of a special class of individuals whose nervous system disposes them to crime from birth; who are born criminals and remain criminals all their lives; who even in childhood display traits which distinguish them from other children. At the same time the writer, while paying a warm tribute to the work done by the Italian school of criminologists, disclaims adherence to the doctrines of the more extreme followers of Professor Lombroso.

So far the work is descriptive rather than controversial, and the author carries his reader with him without raising any serious questioning of the views which he puts forward. In short, the chapters which we have been considering form an excellent

introduction to the study of criminology and will be read with interest and profit by anyone seeking acquaintance with the subject.

When the writer turns to the consideration of the prevention of crime and the treatment of the criminal he enters a much more controversial field, and the reader, who has perhaps accepted without question the conclusions already arrived at as to the nature of crime and the criminal, is astonished to find how far these conclusions are likely to lead when applied to the social conditions at present existing. Not that the author is by any means very extreme in his views as compared with some other criminologists. On the contrary if one once admits that a large proportion of criminals are what they are as a result of innate tendencies for which they are in no way responsible, some such changes in our means of dealing with one of the most difficult of social problems as are indicated by Prof. Kovalevsky would seem to be inevitable. Our present means of dealing with crime Prof. Kovalevsky regards as useless, and worse than useless. In spite of our complicated organisation for the prevention of crime, crimes continue to multiply out of all proportion to the increase of the population. Our prisons only makes men worse. The accidental criminal is converted by them into a professional, and the professional into an incorrigible rascal. They are higher-grade schools for the production of thieves and vagabonds—for whose education society pays. Whether punishment has ever made anyone better is extremely doubtful. In all this there is a good deal of strong assertion, and one would have liked a little fuller proof that serious crime is really increasing so alarmingly. But granting that things are as bad as Professor Kovalevsky asserts what is to be done? Reorganise our whole method of dealing with crime. Let us pay less attention to the nature of the crime and more to the nature of the criminal. Let every one convicted of a criminal offence, no matter how trivial it may seem, be examined by an expert in criminal psychology with the view of determining the treatment suited to the case. For this purpose a sufficient number of lawyers and medical men should be trained in psychology, in mental pathology, and in anthropology. Such students should also receive clinical instruction in the prisons. The judiciary court should consist of an equal number of medical men and lawyers, and convicted prisoners should be sent to establishments somewhat of the nature of asylums in which they would engage in labour and receive instruction specially suited to their case. The duration of detention would depend upon the result of treatment, and liberation would be associated for a time with supervision. Special attention would be given to the careful training of backward, undisciplined, and criminal children. Such, very briefly,

are some of the conclusions at which Professor Kovalevsky has arrived. They are stated with moderation, and supported by argument, and seem worthy of serious consideration. We welcome Professor Kovalevsky's work as a useful addition to an important growing class of literature. The volume is well printed on good paper. It has no index.

W. B. DRUMMOND.

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## Correspondence

### DR FORD ROBERTSON'S REVIEW OF *The Histological Basis of Amentia and Dementia*.

DEAR SIR,—I should be much indebted to you if you would kindly insert the following remarks on the above review of a recent research of mine, which appeared in the April number of the *Review of Neurology and Psychiatry*.

It would not become me, as the author of the paper—and especially so as the review is unfavourable—to make any complaint concerning the opinions expressed by Dr Ford Robertson, though the review generally seems to me, in some of its bald statements, to go somewhat beyond the limits of fair criticism. I would, however, in reply to his strictures, point out that the paper is written to record the results of years of research for the use of workers in psychiatry and not for the education of tyros in the science. My object in the first part of my paper was to prove, not that certain clinical types of insanity exist, not that certain morbid appearances may be found in insanity, but that these two factors are correlated and that a morbid anatomy of insanity exists. It was hence necessary for me to do the whole work myself, and it is beside the question to suggest that my "descriptions of pathological changes . . . with a few trivial exceptions . . . really concern facts with which every asylum pathologist is familiar."

My object in writing this communication is of a different nature. A third of the review is a reply by Dr Ford Robertson to what he appears to consider a direct attack on certain views of his own, and, as will be seen from the following, he cannot have even carefully read the few pages in question before writing his reply.

The following sentence states the point at issue: "That, as suggested by Hill, a considerable amount of blood is held up in this way in the veins and sinuses of the cranium, which would escape on opening the skull-cap in a position in which gravity can act, seems hardly to require proof in view of the important researches which have during the past few years been carried out by this painstaking and experienced physiologist. In view, however, of the opinions expressed in the recent work by Ford Robertson on the 'Pathology of Mental Diseases,' this is not the case, and the writer of the present paper has consequently put the question to experimental proof." Stated in other words, the question at issue is whether or not Hill is right in affirming that "the

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whole blood-content of the brain may change at the moment the pathologist opens the skull."

The first fourteen experiments in my paper prove that 22 c.cs. of *something*, probably blood, is held up. The remaining six, where fluid is admitted into the superior longitudinal sinus, in order that the venous sinuses may remain full during the opening of the dura in the lateral trephine holes, prove that the "something" was blood and *not* subdural fluid, as under these circumstances no recession of the cerebrum occurred. In the paper it is expressly stated (page 496) that "this fact is finally proved by the following series of experiments without which it might be argued that the space was provided by escape of subdural fluid through the foramen magnum."

Dr Robertson, however, says "He assumes that only blood has been displaced. It is certain that also cerebra-spinal fluid has drained away." Dr Robertson's criticism is therefore incorrect and unfair.

The experiments also *do* "indirectly afford the strongest support to the Monro-Kellie doctrine," as the point at issue is simply whether, when the skull-cap is opened (and this *must* be by a trephine hole or some similar means), a recession of the cranial contents occurs, and whether this is from draining away of cerebro-spinal fluid or from escape of held-up blood in the sinuses. If the former, the doctrine of Burrows would receive support, if the latter, the Monro-Kellie. The latter is the one which the experiments support.—I am, sir, yours faithfully,

JOSEPH SHAW BOLTON.

21st April 1903.

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DEAR SIR,—It is easy to understand that from Dr Bolton's point of view my criticisms must seem unduly severe and even unjust. I do not, however, admit that they are really so (saving in respect of possible misinterpretation owing to an unfortunate ambiguity, to which I shall presently refer), when regard is had to the interests involved. The review was not written in any spirit of carping criticism, but in what I believe to be the interests of neurological science and in defence of the rights of other workers, which appear to me to be infringed in consequence of the independence of attitude assumed by the author in dealing with certain pathological questions.

Dr Bolton's word of explanation in reply to my "strictures" has little bearing upon any critical remark in the review, but I would say, in answer to the point raised, that, whatever his purpose may have been, the paper will be read not only by workers in psychiatry, but by very many others who merely seek instruction and information.

I am much surprised that Dr Bolton should adopt the line of defence taken in the next two sentences. I submit that it is untenable. He declares that his object in the first part of his paper was to prove (1) that certain clinical types of insanity and certain morbid appearances found in insanity are correlated, and (2) that a morbid anatomy of insanity exists. The second of these points is additional to what is specifically stated in the paper, but is not of moment here. The first coincides with what is averred in the paper; but far more is

stated in the same place to be included in the subjects dealt with. Dr Bolton says (p. 425)—“The first part of the paper contains the conclusions derived from a careful clinical and pathological study of 200 cases of mental disease,” and “This part of the paper is completed by an account of the etiology and pathology of dementia paralytica.” When we pass from the statement of the programme to enquire as to the manner in which it is carried out, we find that excursions are made far beyond its limits. The very title of the first part—“The Morbid Anatomy of Dementia, together with certain observations on the General Pathology of Mental Disease”—stamps it as covering more ground than is averred in the above letter. It contains subsections upon “The Pathology of Dementia,” “The Pathology of Subdural Deposits,” “The General Pathology of Mental Disease,” “Dementia Paralytica,” as well as others, in which questions of pathogenesis are discussed and descriptions of pathological changes are given which have nothing to do with the question whether certain clinical types of insanity and certain morbid appearances are correlated. In my review I took cognisance of this curious licence that the author has allowed himself, without mentioning it specifically. It is in these intercalated portions of his paper that, by ignoring facts demonstrated by others and propounding theories of pathogenesis that are inconsistent with these facts, he, in my opinion, falls into many errors and must mislead some of those who read simply for instruction. It is to the descriptions of pathological changes given in some of these subsections that I refer in the passage of which a part is quoted by Dr Bolton, and not to anything that is stated in the portions which deal with the correlation of clinical types and morbid appearances. My remark applies, for example, to the descriptions given under the heading of “Morbid Anatomy of Dementia Paralytica” (p. 537 *et seq.*) and in the subsection upon “The Pathology of Subdural Deposits.”

Dr Bolton, by trying to justify his independence of attitude, virtually raises the question of what is the proper method of recording original scientific observations dealing with a subject that has already an extensive literature. It is, however, impossible for me to enter into this question here. Any Continental neurological journal within reach will answer it, and show how a paper written after the manner of the one under discussion departs from a rule that is almost universally adhered to and generally recognised to be necessary, not only in justice to previous workers, but in the interests of the progress of science.

Dr Bolton's charge of incorrectness and unfairness against my criticism of his argument with regard to the displacement of blood from the intracranial cavity on opening the skull, rests upon a mistaken interpretation he has put upon the following passage: “He naturally found that there was evidence that a certain diminution occurred in the volume of the contents of the intracranial cavity when air was admitted. He assumes that only blood has been displaced. It is certain that also cerebro-spinal fluid has drained away.” This passage is interpreted by Dr Bolton as descriptive of his experiments. It really concerns the



bearing of the evidence derived from these experiments upon the "third point," which is under discussion, namely, whether "the whole blood-content of the brain may change at the moment the pathologist opens the skull," an hypothesis which he maintains is proved to be correct by that evidence. In saying that he assumes that only blood has been displaced, I do not allude to his experiments but to what occurs at the moment that the pathologist opens the skull, and I simply make a statement that is consistent with my contention that his experiments have no bearing upon the question whether the whole blood-content of the brain may change at that moment. I freely recognise that the interpretation that Dr Bolton has put upon this passage is one that it is capable of bearing, although my use of the word "air" instead of "water" is inconsistent with it. The ambiguity has arisen from my attempting to deal with a complicated matter briefly. If others have put a similar interpretation upon this passage, I sincerely regret the injustice that has thereby been done Dr Bolton, for which I certainly become responsible.

I gave no details regarding his experiments but merely indicated their nature in a single sentence, because, in my opinion, they do not support either his views upon the general pathogenesis of subdural false membranes or the Monro-Kellie doctrine, and I therefore could not commend them to the attention of the reader. Dr Bolton insists that his experiments do afford the strongest support to the Monro-Kellie doctrine. I cannot agree with this conclusion. The points at issue cannot be settled by arguments drawn from the results of experiments upon the dead subject, such as those that Dr Bolton has carried out. There is the highest authoritative support for this statement. "Experiments on the living animal solely can throw light on these complex questions." These are the words of Dr Leonard Hill, used in reference to the "hydrodynamics of the cranium." The phenomena about which any dispute remains can only manifest themselves whilst the heart is acting, the blood flowing in the cerebral vessels and the cerebral lymph coursing through its special intra-cerebral and extra-cerebral channels.—I am, sir, yours truly,

W. FORD ROBERTSON.

Laboratory of the Scottish Asylums, 23rd April 1903.

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# Review of Neurology and Psychiatry

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## Original Articles

### A CASE OF ACUTE MYELITIS.

By PURVES STEWART, M.A., M.D., M.R.C.P.,  
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*(From the Neuro-Pathological Laboratory, King's College.)*

THE pathological conditions underlying the clinical collection of symptoms known as "acute myelitis" have been much disputed. Widely varying views have been expressed regarding the true nature of the affection. The name myelitis in itself indicates that the disease was originally considered as essentially of inflammatory nature and for a time this view was universally held. Subsequently, however, doubt was thrown upon its truth by the works of numerous observers, notably by Bastian,<sup>1</sup> Pierre Marie,<sup>8</sup> Birch-Hirschfeld,<sup>3</sup> and Bruns,<sup>4</sup> who pointed out that many cases of so-called myelitis are primarily not inflammatory but may be due to thrombosis of one or more spinal arteries. Several of these observers, notably Bastian, go so far as to express grave doubt as to whether inflammation of the cord ever occurs at all, save as a secondary complication of injuries, spinal meningitis or pyæmic processes. Others again, amongst whom may be mentioned Gowers,<sup>6</sup> Ziegler,<sup>14</sup> von Leyden,<sup>7</sup> Oppenheim,<sup>10</sup> Déjérine and Thomas,<sup>5</sup> Schmaus and Sacki,<sup>11</sup> and Nissl,<sup>9</sup> whilst admitting the frequency of thrombotic softening, maintain that myelitis does occur as a true inflammation, primary in the cord itself.

The contradiction between these two views is apparent

rather than real. The confusion which has arisen is due to the fact that similar or almost identical clinical phenomena may be the result of pathological processes of widely different nature. And therefore whilst many cases, probably the majority, of so-called acute myelitis, might perhaps be more accurately designated as thrombotic softening of the cord, yet there remains a certain proportion of cases truly inflammatory in nature and not dependent on any gross arterial obstruction or disease, syphilitic or otherwise.

True myelitis of inflammatory origin is, however, probably less uncommon than would be supposed from the comparatively small number of cases which reach the pathologist's hands. According as the inflammation is in the grey matter or in the white, the result is a poliomyelitis, a leucomyelitis, or, as in the following case, a disseminated myelitis. On the other hand, thrombotic softening of the cord is a more frequently fatal disease, occurring often in syphilitic subjects and presenting clinical features closely resembling those of true inflammatory myelitis.

The following case is an example of true disseminated myelitis.

#### CLINICAL NOTES (FROM KING'S COLLEGE HOSPITAL).

The patient was a lad of 18, a sawyer, whose general health had been good. With the exception of measles in childhood and scarlet fever at the age of 15 he had had no definite illnesses, but was subject to winter cough. There was no history of syphilis, congenital or acquired, nor was there any nervous or mental disease in other members of the patient's family. His mother had rheumatic fever three times and his father was said to have gastric ulcer.

On Dec. 1, 1902, patient took to his bed for four days with what was said to be "influenza," the chief symptoms being pain in the back and legs. From this he appeared to recover and went to work from Dec. 8 till Dec. 11, still complaining, however, of slight pains in the back. On the evening of Dec. 11, on returning from work, he complained of numbness and weakness of the legs and became unable to stand. The numbness spread upwards to the trunk next day and on Dec. 13 retention of urine developed. The bowels were confined from Dec. 10



till his admission to King's College Hospital on Dec. 15 under Dr Ferrier's care.

On examination at the time of admission, he was found to have flaccid paraplegia, complete, of the lower extremities, with absence of knee-jerks and of plantar reflexes. There was retention of urine. Sensation was slightly impaired on the lower extremities and lower part of the trunk and there was a zone of paræsthesia at the level of the 10th costal cartilages. The abdominal muscles and lower intercostals were weak; the upper intercostals, diaphragm and upper extremities were normal. There was no albuminuria. Some general bronchitis was present.

For a fortnight after admission the temperature varied from 102° F. to normal, being of an irregularly intermittent type. On January 1, 1903, slight movement was observed in the left lower limb at all joints. After this date no further notes are available as to the motor, sensory or reflex functions. From January 1 the temperature remained persistently subnormal, 97° or thereabouts, and the patient died on January 4, twenty-four days after the onset of his paraplegic symptoms.

The spinal cord, medulla and pons were sent to the Neuro-pathological Laboratory of King's College for examination.

#### MORBID ANATOMY.

On naked-eye examination the spinal cord appeared normal and there was no localised area of softening. Unfortunately, before being sent on for microscopic examination, the cord had been placed in Müller's fluid. Nissl's stain for the nerve-cells therefore could not be employed. After hardening, sections were prepared by Van Gieson's and Weigert's stains and by Busch's modification of the Marchi method, the latter being in some instances supplemented by counter-staining with picro-fuchsin.

Sections taken from any region of the cord below the cervical showed practically identical appearances at every level, differing only in degree, being more intense in the lower dorsal region than elsewhere. Above the cervical region and in the medulla and pons, the central nervous system was normal.

There was no tract-degeneration, ascending or descending, in the anterior or lateral columns, but in the upper cervical region a few degenerated fibres were visible with the Marchi method,

scantly distributed in the postero-median columns. With that exception, the nerve-elements throughout the cord were remarkably unaffected. Transverse and longitudinal sections showed that nerve-cells, axis-cylinders and medullary sheaths, even within the zones of inflammation, were generally well preserved, or, at the most, merely compressed by the interstitial cell-proliferation about to be described.

On transverse section the appearances of the cord were very characteristic (see Fig. 1). Scattered diffusely over its whole area, in the interstitial tissue both of the white and of the grey matter, were innumerable perivascular collections of mononuclear cells, many of them staining darkly granular with osmic acid. These foci corresponded with the course of the vessels entering all round the periphery of the cord. In arteries which were cut longitudinally, the granular cells formed a dark zone, ensheathing each vessel in its intra-spinal course and occasionally such perivascular infiltration could be traced along the vessel close up to the pia mater. The meninges were healthy.

The individual arteries showed no signs of endarteritis nor were any of them thrombosed. The cellular proliferation was visible:—

1. In the walls of the arteries and capillaries (slightly).
2. In the perivascular sheaths (more abundantly).
3. In the adjacent interstitial substance of the white or grey matter of the cord, the proliferated cells lying between the nerve elements and sometimes compressing them (see Figs. 2 and 3).

No polynuclear leucocytes were present in the foci of infiltration, all the proliferated cells being mononuclear. Closer study of these mononuclear cells showed that they were of two types, one resembling a lymphocyte (of which considerable numbers were present within the vessels) with a large circular nucleus, the other of a connective-tissue type with a less regular, often ovoid nucleus.

Sections stained with Löffler's methylene blue showed staphylococci in considerable numbers, diffusely distributed in small clumps throughout the affected area of the cord. The organisms were visible chiefly in the perivascular lymph-spaces and between the nerve-elements. Some were also present here and there within the mononuclear cells.

This case, with its multiple lesions of vascular distribution,



FIG. 1.

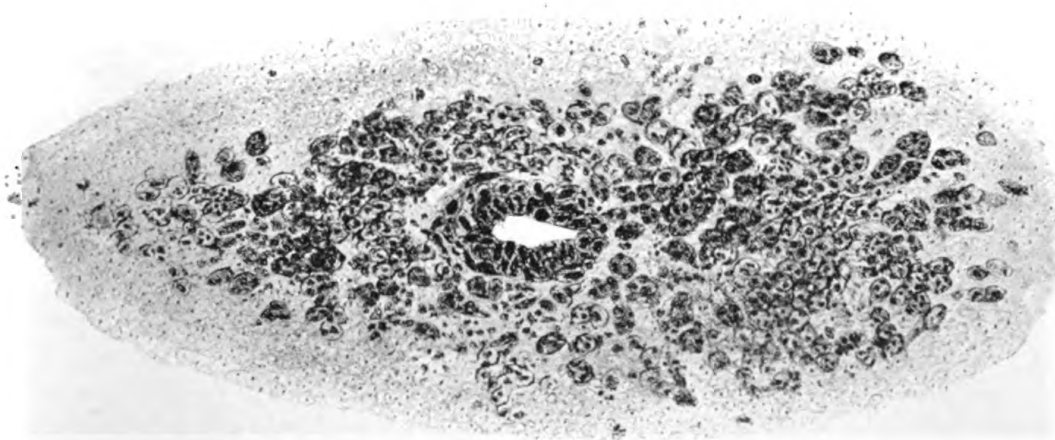


FIG. 2.

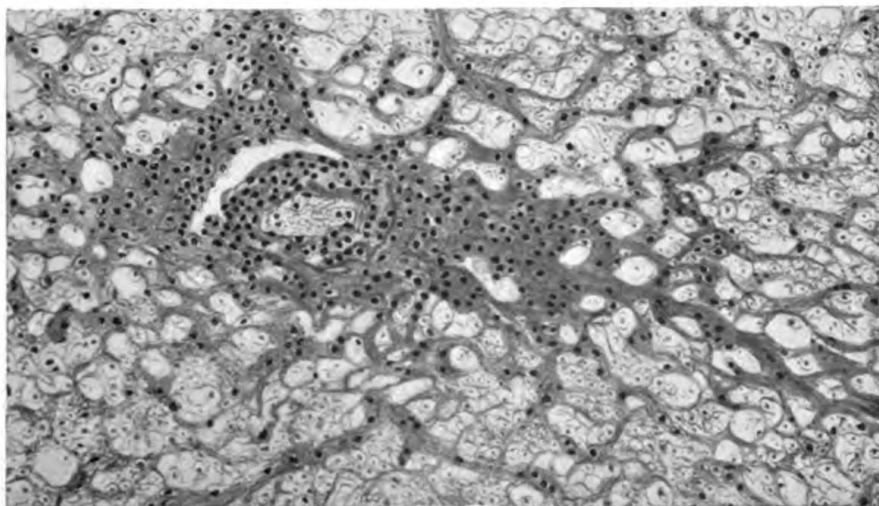


FIG. 3.

FIG. 1.—Transverse section ( $\times 10$ ) of cord in mid-dorsal region. Modified Marchi method. Showing perivascular distribution of inflammatory foci. Note absence of tract degeneration.

FIG. 2.—Section of a focus of perivascular cell-infiltration ( $\times 200$ ). Modified Marchi method. Proliferated cells stained darkly granular by osmic acid.

FIG. 3.—Section of a focus of perivascular infiltration ( $\times 200$ ). Van Gieson stain. Showing proliferation of mononuclear cells in perivascular lymph-space and in interstitial tissue of cord. Nerve elements undegenerated.



is a well-marked example of a true myelitis. The vascular phenomena cannot here be regarded as secondary to preceding degeneration in the nerve elements, for whereas the perivascular infiltrations were of extraordinarily wide distribution, the nerve-fibres and nerve-cells were scarcely affected. The small-celled infiltration was entirely interstitial and only here and there did it succeed in damaging a stray nerve-fibre or cell, hence the scantiness of the tract-degeneration in spite of the enormous number of inflammatory foci. The granular appearance of many of the infiltrating cells was probably a result of the microbic action and not attributable to degenerative products abstracted from the nerve-elements. The granular cells were equally abundant in the foci of infiltration, whether or not the adjacent nerve-elements were degenerated. Nor was the condition the extension of a meningitis; the membranes were healthy. There was no sign of thrombosis of any intra-spinal vessel. Moreover, had the condition been one of thrombosis, there would have been evidence of death of nerve-elements in the thrombosed areas with wide-spread degenerations, ascending and descending, as a result.

The inflammation in this case must undoubtedly be referred to the organisms demonstrated, those having been distributed along the course of the vessels.

Such true inflammation of the cord, as Strümpell has pointed out, may be the result of various exogenous causes, microbic or toxic. In our case, organisms were present in considerable numbers in the affected regions. These organisms exerted their action primarily upon the interstitial tissue of the cord and only secondarily upon the nerve elements.

Gross necrotic changes, such as those resulting from thrombosis of a spinal artery, belong to quite a different category and the pathological appearances are unlike those present in our case.

Thrombotic softening of the cord, with its destruction *en masse* of nerve elements within the area affected, and its consequent ascending and descending degenerations, is commonly due to coarse vascular disease and should be clearly distinguished from the multiple interstitial lesions of a case like our own. Singer<sup>12</sup> in a recent paper appears inclined to throw doubt upon the existence of true myelitis except as a secondary complication of inflammatory disease elsewhere. Such a view is hardly justified by the facts,

still less is it correct to affirm, as that writer does, that "the absence of small-celled infiltration is a striking feature in all records," for Bielschowsky,<sup>2</sup> Gowers,<sup>6</sup> Oppenheim,<sup>10</sup> Schmaus-Sacki<sup>11</sup> and others have described and figured cases closely resembling the present one.

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### NOTE OF A CASE OF JUVENILE GENERAL PARALYSIS OF INSANE.

By JAMES CAMERON, M.B. Lond., M.D. Edin.

W. D., a short, thick-set lad of 14½ years of age, came before me at New Town Dispensary, March 22, 1898.

The statement of his mother was that during the year previous, patient's memory, until then fair, had become noticeably defective, so much so that although he had reached the Fourth Standard at school he had to be discharged on this account. Further, he was said to be becoming abnormally shy and had taken to stuffing his pockets with collections of rubbish. He was stunted, plump, stupid, shy, answered in monosyllables, was tearful and easily coerced by the younger members of his family, unsteady on his limbs and had much increased reflexes.

The testicles were undescended. The case was entered as "Mental Deficiency," and he was advised to be taken daily with his father, a quarryman, to his work in the country. I have since learned from the Infirmary Registrar that previously to my

seeing him, patient had been an inmate of Ward VI., Royal Infirmary, from January 5 to January 10, 1898, his case being entered as "Arrested Mental Development."

Patient next seems to have developed maniacal symptoms, shouting at night and using forcible language which led, as I afterwards learned, to his being sent to Morningside Asylum, where he remained from September 2, 1898, to November 12, 1898. Dr MacRae, the Assistant Physician there, kindly informs me that the mental symptoms were much as above and that one of the last entries takes cognisance of development of muscular weakness and unsteadiness of gait.

On patient's coming under my care some months after this last date, the dementia was more marked, the weakness increased, the reflexes notably increased, the gait unsteady and patient continually rubbed his head in a dreamy fashion.

About two and a half years before death a severe attack of chorea occurred, during which arsenic was given, and on this abating, the diagnosis of G. P. was definitely made.

Two years before death, patient became bed-ridden, emaciated and demented, the right pupil being generally dilated, while noisy nights, dirty habits and bed-sores completed the ordinary picture of G. P.

Definite delusions were not noticeable. The discs were unable to be examined from patient's nervousness.

Death occurred 25th October 1902, at age of about 19½ years, after several days of severe and continued "congestive attacks" with the usual high temperature (105° F.).

A family history of syphilis was not elicited, but a younger brother has "specific teeth."

Persistent treatment by iodide of potash and mercury had little effect, although life was doubtless prolonged by cod liver oil and careful nursing.

The brain was handed over to Dr Ford Robertson for examination and report.

#### *Pathological Report.*

The examination of the brain fully confirms the clinical diagnosis of general paralysis. The organ, after hardening in formalin, weighs 35 ounces. It shows well marked general atrophy. The pia-arachnoid is everywhere milky, thickened and

finely granular on its surface. The arteries show slight general thickening of their walls. On section the organ is seen to be much congested. The cortex is everywhere narrow and of an abnormally dark tint. The white matter and basal ganglia have a somewhat shrunken appearance. Over a considerable area of the inferior aspect of both temporo-sphenoidal lobes, the morbid process has gone on to actual softening, affecting especially the deeper layers of the cortex. There are no other focal lesions.

Sections from two portions of the right motor frontal region show well marked sclerosis of the first layer of the cortex and of the white matter, great hypertrophy and proliferation of neuroglia in the deepest layer of the cortex and in the adjacent white matter, and occasional small areas of sclerosis in the other cortical layers. Most of the vessels show fibroid thickening of their walls and irregular dilatation of their lumina. The nerve cells are extremely degenerated. Many can be seen in the last stages of disintegration. They are greatly diminished in numbers.

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### **A CONTRIBUTION TO THE SPINAL ROOT LOCALISATION OF THE KNEE-JERK, ACHILLIS-JERK AND PLANTAR REFLEX.**

By EDWIN BRAMWELL, M.B., M.R.C.P.

THE following observations, which refer to a case of very early preataxic tabes, appear to afford definite evidence regarding the spinal root localisation of the knee- and Achillis-jerks.

#### **CLINICAL REPORT.**

J. M., a cellarman, aged fifty-seven, was admitted to the Edinburgh Royal Infirmary, under the care of Dr Byrom Bramwell, on January 28th, 1902, suffering from symptoms of intrathoracic pressure. It is unnecessary to enter into detail regarding these symptoms. The clinical history of the case has been recorded in full by Dr Byrom Bramwell.\*

The points of importance in relation to the question at present under consideration are as follows:—

At the age of thirty-seven the patient had had rheumatic fever. Ever since he had been troubled with pains in the legs

\* "Clinical Studies," by Byrom Bramwell, M.D., 1903, vol. i. part ii. p. 130.



from time to time. It seemed probable that these were "rheumatic pains," and not the lightning pains of tabes.

He had been treated in the Lock Wards of the Royal Infirmary for *syphilis*, a chancre followed by secondary symptoms, in 1894.

Upon examination, it was found that the pupils were unequal, the right measuring 3 mm., the left 2 mm. The inequality of the pupils was very possibly, however, a pressure symptom produced by the intrathoracic tumour, and cannot be insisted upon as a point of any importance in relation to the tabetic condition. *The reaction of both pupils to light was distinctly sluggish*; they contracted well on convergence.

*Both Achillis-jerks and the left knee-jerk were absent, the right knee-jerk being active.* Reinforcement was employed, and the observation confirmed on several different occasions. Apart from the above mentioned signs, a careful examination elicited no further evidence of tabes. There was no ataxia and no Rombergism. No anæsthesia or analgesia was detected on the arms, trunk, or lower limbs. The patient stated that there had been no incontinence of urine, and that there was no delay in commencing the act of micturition. The arm-jerks were present on both sides. *The plantar reflexes were present*, and of the flexor type. The cremasteric reflexes were not examined. No special entry is made in the notes as to the sexual functions, the state of the muscle tone, or the condition of the muscular and articular senses in the lower limbs.

On the night of March 11th, the patient was seized with an attack of difficulty of breathing and faintness which proved fatal.

#### PATHOLOGICAL REPORT.

The post-mortem examination was made on the day following death. A large new growth was found involving the bronchial glands and the root of the left lung, and extending along the bronchi into the lung tissue.

The spinal cord below the level of the third cervical segment was obtained for examination.

With the naked eye nothing abnormal was noticed.

The spinal cord was prepared for microscopic examination.

*Method.*—Within half-an-hour after its removal from the body the cord was placed in ten per cent. formalin.

After two days it was transferred to Müller's fluid, where it remained for some months prior to examination, the fluid being occasionally renewed.

After hardening, the cord was divided into segments by a series of transverse incisions between successive roots. Each segment was then divided by another transverse incision into an upper and a lower half.

The upper halves of successive segments were retained in series by means of a horse-hair passed through the right anterior horn from above downwards (Series A). The lower halves of the segments were threaded in an exactly similar manner (Series B). In the case of the longer segments only a part of each half segment was taken, *i.e.* the portion lying nearest the centre of the segment.

Series A was placed for six days in fresh Busch's fluid, the fluid being changed after three days.

Both series were dehydrated and prepared for cutting in photoxylin in the usual manner. Each piece of cord was mounted on a numbered block. In the case of Series A, the upper surface of each piece was placed next the block, whereas in Series B the lower surface rested on the block. In this way it will be seen that the first sections taken from each block represented those lying nearest the centre of each spinal segment.

The hole made by the horse-hair through each right anterior horn served to identify the upper and lower surfaces of individual sections.

Sections from twelve different levels, prepared by Busch's method, were examined.

Sections were cut from each segment and stained by Ford Robertson's modification of Heller's method.\*

A few sections from different levels were stained by Van Gieson's method.

Nissl's method was not available, since the whole cord had been hardened in Muller's fluid.

#### *Results of Microscopical Examination.*

In the sections prepared by Busch's method, no degeneration was met with.

\* "A Modification of Heller's Method of Staining Medullated Nerve-Fibres," by W. Ford Robertson, *Brit. Med. Journ.*, March 18, 1897.

All the sections stained by the modified Heller method, down as far as the level of the first sacral segment, showed a distinct degeneration in the posterior columns.

Van Gieson preparations demonstrated an increase of the interstitial tissue in the degenerated areas.

*Detailed description of the position of the degenerated fibres at different levels.*

No degenerated fibres are to be seen in any of the sections below the level of S. 1. A section at the level of S. 2 is shown in Plate 9.

S. 1.—To the inner side of the right posterior horn lying in contact with the grey matter, in the position of the "root entry zone" of Strümpell and Westphal, a pale area is distinctly visible to the naked eye. Under the microscope many of the nerve fibres in this area are seen to be much reduced in size and they are more widely separated from each other than is the case in S. 2. There is a marked atrophy of the right posterior root at this level. The network of fibres in the posterior horn is less dense than on the opposite side. The contrast between the degenerated posterior root on the right side and the healthy root on the left is very striking. The left posterior root appears quite healthy to the naked eye; under the microscope, however, a few degenerated fibres are to be seen in the root entry zone on the left side. Sections from the lower part of this segment show no evidence of degeneration on either side.

L. V.—The degenerated area on the right side extends further inwards towards the median fissure than in the last segment. A posterior root, many of the fibres of which are degenerated, may be seen insinuating itself between the grey matter and the degenerated area to the inner side of the right posterior horn. On the left side, the fibres of the posterior root are profoundly atrophied, and the pale root entry zone is now distinctly visible to the unaided eye. The region occupied by the degenerated fibres is more extensive on the right than on the left side.

L. IV.—At this level the area of degeneration on the right side lies nearer the postero-median fissure, and is distinctly

separated from the posterior horn by a layer of perfectly healthy fibres. No atrophic fibres are seen in the right posterior root.

On the left side the pale area still lies in contact with the grey matter of the posterior horn. The posterior root is very markedly degenerated. The network of fibres in the posterior horn is distinctly less dense than on the opposite side.

*L. III.*—The degenerated area on the right side lies nearer the middle line. The fibres of the root entry zone, as in the last section, are quite healthy on this side.

The area occupied by the degenerated fibres has increased in size on the left side. A thin band of healthy fibres lies between it and the posterior horn. A few degenerated entering fibres are still, however, to be seen in the root entry zone in sections from the lower part of this segment.

Above this level no degenerated fibres were seen entering the cord.

The degenerated fibres as they ascend gradually approach the middle line until in the cervical region they form a narrow strip lying on each side of the postero-median fissure.

In several of the photographs which illustrate this article Lissauer's tract is noticed to be unduly pale. The fibres of the tract in the normal cord are smaller and more widely separated in this region than elsewhere, and it was extremely difficult to say whether there was actual degeneration in this tract or not.

SUMMARY OF THE OBSERVATIONS UPON WHICH THE CONCLUSIONS  
ARRIVED AT IN THIS PAPER ARE BASED.

Before stating conclusions, it will be well to summarise our observations in tabular form.

<i>Table showing the condition of the Knee-jerks, Achillis-jerks and Plantar Reflexes in the case of J. M.</i>			
		LEFT.	RIGHT.
Knee-jerk . . .		absent	active
Achillis-jerk . . .		absent	absent
Plantar reflex . . .		present	present

*Table showing the Posterior Roots degenerated in the case of J. M.*

					LEFT.	RIGHT.
L.	2.	.	.	.	0	0
L.	3.	.	.	.	slight	0
L.	4.	.	.	.	marked	0
L.	5.	.	.	.	marked	slight
S.	1.	.	.	.	slight	marked
S.	2.	.	.	.	0	0

The accuracy of the clinical observations is beyond doubt, they were confirmed on several separate occasions.

The method employed in determining the individual spinal segments has been already described. It is interesting to compare the shape of the grey matter at different levels with Dr Alexander Bruce's illustrations of the normal spinal cord.\* Outline drawings taken from Dr Bruce's work have been placed opposite the photographs illustrating this paper, since they afford additional corroborative evidence of the exact level from which the various sections were taken.

#### CONCLUSIONS.

1. *Loss of the Achilles-jerk may result from a lesion limited to the fifth lumbar and first sacral posterior roots.*
2. *Loss of the knee-jerk may result from a lesion confined to the fourth and third lumbar posterior roots.*
3. *The afferent path of the reflex arc for the plantar reflex in all probability enters the cord by a posterior root or roots situated below the level of the first sacral.*

The author's thanks are due to Drs Alexander Bruce and Byrom Bramwell, who have kindly examined the sections and confirmed his observations.

\* A Topographical Atlas of the Spinal Cord, by Alexander Bruce, M.D. Williams and Norgate, 1901.

TABLE SHOWING THE OPINION OF VARIOUS AUTHORS AS TO THE  
SPINAL SEGMENTAL LOCALISATION OF THE KNEE-JERK,  
ACHILLIS-JERK AND PLANTAR REFLEX.

## KNEE-JERK.

	Dejerine. <sup>1</sup>	Edinger. <sup>2</sup>	Gowers. <sup>3</sup>	Horsley. <sup>4</sup>	Leyden and Goldscheider. <sup>5</sup>	Oppenheim. <sup>6</sup>	Peterson. <sup>7</sup>	Russell, Batten and Collier. <sup>8</sup>	Sherrington. <sup>9</sup>	Starr. <sup>10</sup>
L. 2	...	1	1	...	1	1	...	...	1	1
3	1	1	1	1	1	1	1	1	1	...
4	...	1	1	1	1	1	...	...	1	...

## ACHILLIS-JERK.\*

L. 5	1	...	1	1	...	1	1	...	...	...
S. 1	1	...	1	...	1	1	...	1	1	...
2	...	...	...	...	1	...	...	...	1	...
3	...	1	...	...	...	...	...	...	...	1
4	...	1	...	...	...	...	...	...	...	1
5	...	1	...	...	...	...	...	...	...	1

## PLANTAR REFLEX.

L. 5	...	...	...	...	...	...	1	...	...	...
S. 1	...	1	1	1	1	1	1	...	...	1
2	...	1	1	1	1	1	1	1	...	1
3	...	...	...	...	...	...	...	...	...	...
4	...	...	...	...	...	...	...	...	...	...
5	...	...	...	...	...	...	...	...	...	...

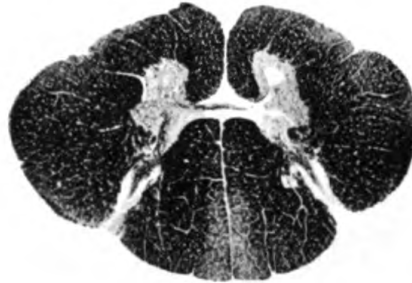
\* The observations of Gowers, Horsley, Peterson, Russell, Batten and Collier and Sherrington refer to ankle clonus.

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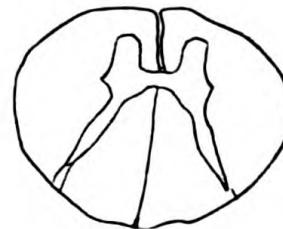
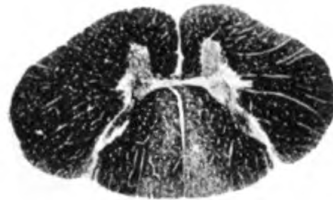
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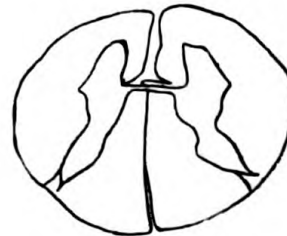
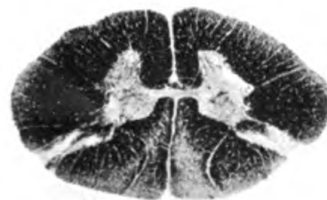
PLATE 8.



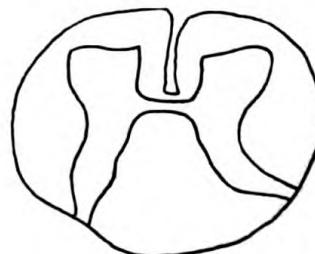
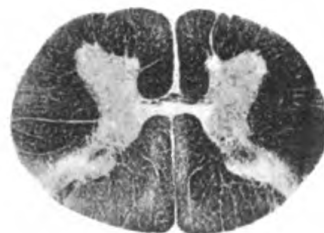
C. 4.



D. 8.



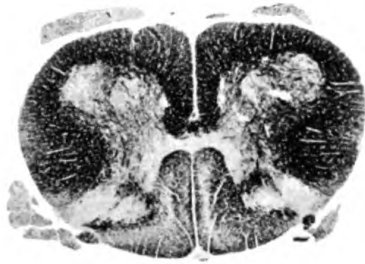
L. 1.



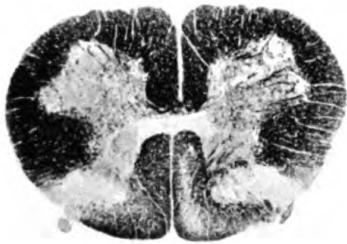
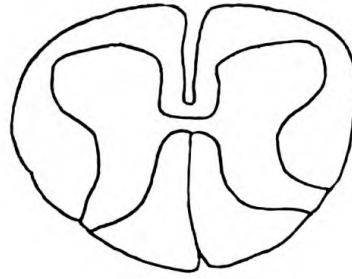
L. 2.

Photographs of the spinal cord from a case of preataxic tabes in which both Achilles-jerks and the left knee-jerk were absent, the right knee-jerk being active. The plantar reflexes were present. Opposite each photograph is a reduced outline tracing of each segment taken from Dr Alexander Bruce's Topographical Atlas of the Spinal Cord.

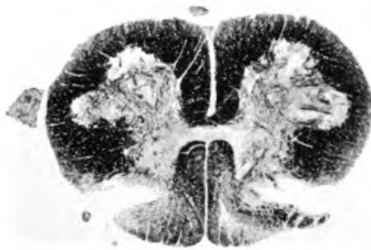
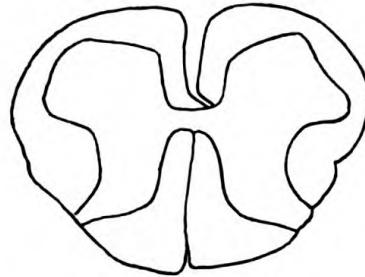




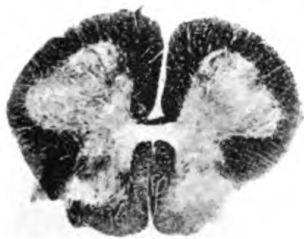
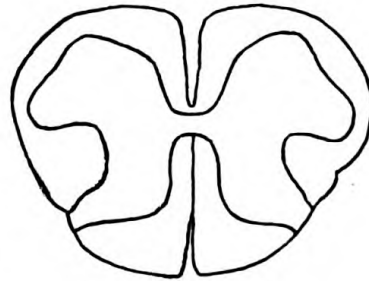
L. 3.



L. 4.



L. 5.



S. 1.



S. 2.





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## Abstracts

### ANATOMY.

#### **THE POSTORBITAL LIMBUS; A FORMATION OCCASIONALLY (213) MET WITH AT THE BASE OF THE HUMAN BRAIN.**

E. A. SPITZKA, *Philad. Med. Journ.*, April 11, 1903.

THIS name is given by S. to a peculiar formation on the orbital surface of the frontal lobe, consisting of a "curved, welt-shaped eminence, demarcated from the rest of the orbital region by a more or less distinct incisure," and apparently due to a growth of this part of the brain downwards round the lesser wing of the sphenoid, into the middle cranial fossa.

S. has found it to occur in the brains of a native of Japan, two professors and an American scientist, and notes its presence in figures of the brain by Retzius, and in Wagner's plate of the brain of Gauss.

Further observations are required to determine the frequency of occurrence and the significance of this formation.

DAVID WATERSTON.

#### **OBSERVATIONS ON THE RELATIONS OF THE DEEPER PARTS (214) OF THE BRAIN TO THE SURFACE.** JOHNSON SYMINGTON,

*Journ. Anat. and Physiol.*, vol. xxxvii. p. 241.

IN this paper S. gives the results of his examination of the head of an adult female which was hardened by formalin, frozen, and sectioned by a method previously described. In the plates the author figures the head, viewed from the side, with the outline of the mesial aspect of the brain marked on the surface, figures also the relation of the ventricular cavities to the lateral aspect of the cranium, and gives photographs of several horizontal sections of the skull and brain.

The care with which the relationships have been worked out render the plates of value for reference on the subject. One is especially struck by the demonstration of the vertical character of the long axis of the medulla and pons, and by the corresponding course from the motor area of the pyramidal fibres, which pass even slightly forward from their origin in the cortex to the point where they enter the pons.

The course of the visual fibres in relation to the side of the head is also figured.

The author compares his results with others previously published, and criticises several points of difference.

DAVID WATERSTON.

**ON THE PALLIO-TECTAL OR CORTICO-MESENCEPHALIC  
(215) SYSTEM OF FIBRES.** C. E. BEEVOR and Sir VICTOR  
HORSLEY, *Brain*, Winter 1902, p. 436.

In this investigation localised lesions were made in various regions of the cortex cerebri, in the monkey, cat, dog and badger; the animals were killed at the end of three weeks, the brain in each case was stained by Busch's modification of the Marchi method, and cut in serial sections.

In the cases (cat and monkey) in which lesions were made on the external surface of the *occipital cortex*, a considerable number of degenerated fibres could be traced to the mesencephalon. Those going to the superior colliculus (ant. or sup. corp. quad.) were large and stood out distinctly from the medium-sized occipito-thalamic and occipito-geniculate fibres, and from the small callosal fibres. They were distributed to the whole breadth of the stratum griseum profundum of the anterior colliculus.

When the *temporal lobe* was involved, in the cat fibres were traced to the optic thalamus, internal geniculate body, posterior brachium of the corpora quadrigemina and the pontine nucleus. A number of fibres in the posterior brachium turned up towards the posterior colliculus but could not be followed into that ganglion. The fibres to the pons passed downwards on the outside of the crus and ended in the upper and outer part of the frontal extremity of the pontine nucleus; they could not be traced farther caudalwards than the junction of the upper and middle thirds of the pons. In the monkey, in which the lesions were confined to the posterior half of the particular gyrus operated on, degenerated fibres were followed to the optic thalamus, the corpus geniculatum internum, the posterior brachium and the locus niger.

In the *motor cortex*, lesions were made in the monkey, cat, dog and badger. In the monkey the system of fibres connecting the excitable cortex with the midbrain was only slightly developed. In the dog no such fibres were seen at all, but as only a single

animal was examined the authors 'do not regard the question of their presence or absence in the dog as settled. As a matter of fact, Muratoff\* in 1893 in the dog described fibres, very few in number, leaving the pyramidal tract in the crusta and passing to the tectum. *In the cat and badger* a very well marked system of pallio-mesencephalic fibres was present. These fibres were found to leave the pyramidal tract in two definite groups—an upper and a lower. The fibres of the upper group became detached from the internal capsule, passed backwards through the thalamus and ended partly in that body and partly in the anterior and posterior colliculi. The fibres of the lower or cruro-tectal group arose from the crus cerebri at points all along the outer third of its dorsal surface and were traced to the locus niger and to the anterior and posterior colliculi. In four cats the lesion involved the *whole of the fronto-parietal region*, and in each case this led to the degeneration of a large number of pallio-mesencephalic fibres, and also to extensive terminal degeneration in the optic thalamus. This confirms the observations of Boyce and others, that, in the cat especially, the motor cortex is in close association with the anterior colliculus.

When the lesions were confined to the *non-excitabile part of the frontal lobe* in monkeys, and to the frontal pole in cats, there was very striking degeneration of fronto-thalamic fibres, but the only mesencephalic centre to which fibres could be followed was the upper or anterior part of the locus niger. No fibres were found going from the non-excitabile part of the frontal lobe to the tectum.

Making a general statement, the authors say: "From the frontal cortex no fibres could be traced by us to the mesencephalon; from the temporo-sphenoidal cortex very few fibres; while from the occipital lobe a fair number, and from the excitable cortex a large number of fibres can be followed to the corpora quadrigemina and mesencephalon." SUTHERLAND SIMPSON.

**THE DIRECT VENTRO-LATERAL PYRAMIDAL TRACT.** WILLIAM (216) G. SPILLER, *Neurolog. Centralbl.*, 1902, No. 12, p. 534.

IN this short note the author reviews the work which has been published on this subject. It will be remembered that Spiller in 1899 traced a tract of degenerated fibres from the pons varolii to the first cervical segment of the cord, fibres which appeared to him to have split off from the pyramidal tract in the mid-pontine region and to have descended to occupy a ventro-lateral position in the cervical cord. He then thought that these fibres arose either in the cortex or basal ganglia and constituted a constant tract to which, however, he did not give any name. In 1901, I

\* Muratoff, *Archiv. f. Anat. u. Physiol.* 1893, Heft 3 und 4, S. 97.

published in *Brain* four cases of degeneration of what was evidently this set of fibres; I was able to trace them from the internal capsule to the lumbar region of the cord, and having proved that they were a part of the pyramidal system suggested that the name "Ventro-lateral Pyramidal Tract" should be given to them. Spiller takes exception to this designation, since this name only defines the position which the tract occupies in the spinal cord, and takes no account of the fibres running in the pons and medulla. He offers, however, no alternative name, but wishes to prefix "direct" to the title I suggested, in order to clearly indicate that it is an uncrossed tract. Seeing that there is no similar crossed tract it seems to me hardly necessary to lengthen the title of this bundle of fibres, nor do I think that another title can be found expressing its origin and main distribution more clearly and shortly than the one I first used.

Spiller agrees with me that these fibres have no connection with Helweg's "Dreikantenbahn" (the Fasciculus periolivaris of v. Bectereu), and that the degeneration seen by Mott and Tredgold (*Brain*, 1900) and Dejerine (1900) are other instances of a ventro-lateral pyramidal tract. He thinks that the fibres described by Purves Stewart (*Brain*, 1900) are a further example of the same tract; I am convinced that this is not so, as Stewart's tract "x" has neither the shape nor position of the ventro-lateral pyramidal tract.

STANLEY BARNES.

**A PHYSIOLOGICAL, ANATOMICAL, AND PATHOLOGICAL  
(217) STUDY OF THE GLOSSOPHARYNGEUS AND VAGUS  
NERVES IN A CASE OF FRACTURE OF THE BASE OF  
THE SKULL.** W. G. SPILLER, *Univ. Pennsylvania Med. Bull.*,  
March 1903.

A MAN of 54 fell from a height of ten feet on his head, but after being unconscious for twenty minutes he was able to get up and walk. No loss of motor power could then be detected in his limbs, and sensation and the various reflexes remained normal, but he was quite unable to swallow, and his tongue deviated to the left side. Later, on examination, the tongue was protruded straight, and the face was normal, but the left side of the soft palate moved deficiently, and the left half of the larynx was completely paralysed. Taste was equally acute on each anterior two-thirds of the tongue.

Swallowing was so severely affected that it was only possible to give him nourishment per rectum, but despite this he died on the 45th day.

At the autopsy a fracture of the left side of the occipital bone was found, and from the line of fracture a spicule of bone projected upwards, lacerating the under surface of the left lobe of the

cerebellum and the glossopharyngeal and vagus nerves of the same side. The peripheral portions of these nerves were severely degenerated, but the other cranial nerves were normal. The tracts found degenerated in the medulla by Marchi's method included the descending roots of the 9th and 10th nerves, fibres of these nerves passing to the nucleus ambiguus, others across the raphé to the right fillet, and finally some to the dorsal glossopharyngeal-vagus nucleus. The cells of the latter nucleus as well as those of the nucleus ambiguus were pathologically changed, and had excentric nuclei. Degenerated muscle fibres were visible in the left laryngeal, pharyngeal, and soft palate muscles. The only important visceral change was the presence in both lungs, but chiefly in the left, of areas of broncho-pneumonia and of multiple small abscesses.

One of the most interesting points in this case was the persistence of the sense of taste on the anterior two-thirds of the tongue, which clearly shows that the taste fibres of this distribution do not enter the medulla in the glossopharyngeus root. Unhappily the condition of this sense on each half of the posterior third of the tongue is not recorded, but the circumvallate papillæ of the left side were found degenerated.

Insomuch as the pharyngeal reflex was preserved on the side of the injured 9th-10th nerves, the sensory supply of this part cannot be confined to these nerves, while the only slight weakness of the left side of the soft palate points to its innervation being only in part through the glossopharyngeus. The lung changes are attributed to paralysis of the vagus, and regarded as partly trophic and not entirely secondary to the laryngeal paresis and the consequent entrance of saliva, etc. A slightly accelerated pulse-rate was the only abnormal cardiac symptom.

The cell changes found in it are taken as a proof that the dorsal 9th-10th nucleus is motor.

GORDON HOLMES.

**THE SPINAL ORIGIN OF THE CERVICAL SYMPATHETIC (218) NERVE.** PERCY T. HERRING, *Journ. Physiol.*, vol. xxix. p. 282.

GASKELL first pointed out that the lateral horns of grey matter in the thoracic region of the spinal cord contained cells which probably gave rise to the motor nerves of the vascular and glandular systems. Mott and Sherrington supported the same view. Other observers by dividing the cervical sympathetic and studying the cord for Nissl degeneration of the cells of origin have obtained results partly confirmatory, partly contradictory. Recently Anderson\* after dividing the cervical sympathetic in kittens, found a

\* *Journ. Physiol.* xxviii. p. 499.

lack of development of the lateral horn of grey matter in the upper thoracic region of the cord on the same side as the lesion.

The results embodied in this paper are confirmatory of Anderson's. The cervical sympathetic of one side was divided in adult cats and in one kitten. After a period of from two to four weeks the animals were killed and the cords examined by Nissl's method. Chromatolysis was found in the cells of the lateral horn of the same side as the lesion, beginning above at the level of emergence of the 8th cervical nerve, most marked in the 1st, 2nd and 3rd thoracic segments, the affected cells diminishing in number down to the 6th thoracic segment. Changes were not found in any other part of the spinal cord. The cells are small, multipolar, and often compressed from side to side so as to appear spindle-shaped; they spread outwards for a considerable distance into the white matter of the lateral columns.

In the kitten the cells in the lateral horn of the injured side were far fewer and smaller than the cells in the corresponding situation on the healthy side, especially in the 1st, 2nd, and 3rd thoracic segments.

AUTHOR'S ABSTRACT.

**THE COMPARATIVE ANATOMY OF THE NERVUS ACUSTICUS.** (219) GORDON M. HOLMES, *Trans Roy. Irish Acad.*, vol. xxxii. p. 101.

IN this paper the author traces the development and differentiation of the nuclei associated with the auditory nerve. He points out that the cochlea, as such, appears late in the animal scale; in the Cyclostomata the utricle and saccule are simple, uncomplicated structures; in the Teleostei the utricle shows the germ of a cochlea, in the shape of a small diverticulum, the lagena; in the Amphibia the lagena attains larger size; it varies greatly in the amount of its development in the Reptilia, being largest in the Crocodilia; in Birds it resembles the latter group; in Mammals the spiral rolling of the lagena gives rise to a true cochlea, but in the Monotremes it is hardly more developed than in the Reptiles. Holmes begins his comparative study with the Amphibia, owing to the great complexity of the medulla of Fishes; merely noting that in the latter the auditory fibres end chiefly around the cells of a large tuberculum acusticum, while many enter the cerebellum directly and others descend into the spinal cord, constituting a descending vestibular root.

*Amphibia.*—*Rana* and *Salamandra* are taken as types. The auditory nerve is separated here into dorsal and ventral roots. The former is distributed to the lagena, the papilla basilaris, the ampulla of the posterior semicircular canal, and macula neglecta



of the saccule; the latter to the rest of the saccule, the utricle and the anterior and posterior ampullæ. The dorsal root ends in a large nucleus just below the lateral margin of the medulla. From this dorsal nucleus a tract of fibres arises which passes ventralwards, lateral to the descending root of the fifth nerve, to cross the raphe to the opposite side, thus closely resembling the corpus trapezoides of higher types; the termination of this strand in the ganglion posterius mesencephali (posterior quadrigeminal body) supports the view that this is a secondary cochlear tract. A second nucleus lies ventral to the one just described and consists of much larger cells; to this the fibres of the ventral root chiefly pass. Both roots also furnish fibres directly to the cerebellum. Although there is no absolute separation of the nerve cells for the cochlear fibres here, it seems probable that the rudiments of the cochlear nuclei are contained in the dorsal nucleus, together with the cells corresponding to the nucleus dorsalis vestibuli. The larger cells of the ventral nucleus represent Deiters' nucleus, and can easily be seen to furnish a tract to the spinal cord.

*Reptilia.*—The type selected for full description is the Alligator. Three nuclei are associated with the auditory nerve, which in this case enters the medulla by a single root, many fibres passing directly to the cerebellum. The nucleus dorsalis is situated in the dorsal margin of the medulla and is folded on itself at its caudal extremity, so that the appearance of two distinct nuclei is met with in some sections; it extends forwards to a point much anterior to the level of entrance of the auditory root. The nucleus laminaris is best developed at the point of entrance of the auditory nerve, but extends for some distance above and below this level. It lies ventro-mesial to the nucleus dorsalis, and by its presence causes the eminentia acustica to project considerably into the cavity of the fourth ventricle. The nucleus ventralis, formed of large scattered cells, lies just ventral to the nucleus laminaris; when stained by Nissl's method its cells are seen to belong to the motor type. By studying the central connections of these nuclei their homologies can to a large extent be established. From the nucleus dorsalis a strand of fibres arises which runs ventralwards, parallel with the margin of the medulla, to reach a group of cells lying close to the ventral aspect of the medulla; here many of these fibres end but others pass on to the similar nucleus of the opposite side. These two ventral nuclei represent the superior olives. The strand of fibres just described forms, then, a secondary cochlear tract (corpus trapezoides); and from the region of the superior olive another strand, tertiary cochlear tract (lateral fillet), can be followed to the region of the nucleus posterior (posterior quadrigeminal body). For these reasons the nucleus dorsalis may fairly be regarded as the terminal centre for the

cochlear fibres. As regards the nuclei laminaris and ventralis, a large bundle is furnished from both of these to pass to the cerebellum on the same side; also a large commissural strand can be traced through the raphe to the opposite nuclei and to some extent to the cerebellum; further fibres enter the posterior longitudinal bundle. From the large cells of the nucleus ventralis other fibres arise which constitute a special spinal tract. So, then, the nucleus laminaris and nucleus ventralis are vestibular nuclei, and the large cells of the latter represent Deiters' nucleus. Holmes goes on to describe more briefly the conditions in various other members of the Reptilia; the chief difference found is in the amount of development of the nucleus dorsalis, which varies according to the condition of the cochlea. The vestibular nuclei remain almost constant in all members of this class.

*Aves.*—Columba is taken as the type. The auditory nerve here enters the medulla by two roots; of these the anterior is usually regarded as the vestibular and the posterior as the cochlear portion. Holmes shows that this is not quite a correct description, as the nerve fibres from the posterior semicircular canal join the posterior root. The anterior, or vestibular, root sends many fibres directly to the cerebellum; while many others turn downwards and end around cells which constitute a descending vestibular nucleus; yet others end around a nucleus of large cells just ventral to the cerebellum (Deiters' nucleus); and, lastly, others pass to a large cell mass situated just below the floor of the fourth ventricle (nucleus dorsalis). All these nuclei communicate with those of the opposite side by a commissural system of fibres. The posterior, or cochlear, root is associated with three nuclei: the nucleus magnocellularis, lying just dorsal to the anterior extremity of the posterior column nuclei; the nucleus angularis, which appears at the level of entrance of the posterior root; and the nucleus laminaris interposed between these two. No fibres from the vestibular nerve can be traced to these nuclei. The relations of the nucleus laminaris are peculiar; it furnishes a large strand to the cerebellum, and also to the commissural system passing to the opposite side; Holmes explains this by regarding it as the centre to which the fibres from the posterior semicircular canal (*i.e.* vestibular fibres) pass. From the nucleus magnocellularis and nucleus angularis a tract of fibres arises which reproduces the course of the corpus trapezoides as previously described. This tract comes into relation to the superior olive, and from this level a tertiary cochlear tract passes upwards to end in the ganglion laterale mesencephali (posterior corpus quadrigeminum). These nuclei, then, must be regarded as the central cochlear nuclei; while the nucleus laminaris is to be looked on as a specialised part of the vestibular apparatus.

*Monotremata*.—No members of this group were examined.

*Marsupialia*.—*Macropus* is the selected type. The auditory nerve here again enters the medulla by a single root, so that the homologies of the various nuclei associated with it can only be established by an examination of their central connections. Two large nuclei, one dorso-lateral, the other ventro-lateral in position, are found to give rise to bundles of fibres which sweep downwards and forwards to form an undoubted corpus trapezoides. The fibres from this come into relation to a nucleus lying ventral to the facial nucleus (superior olive); from this a further strand passes brainwards. Another tract of fibres connects these auditory nuclei with the superior olive; this appears to represent the auditory striae. Owing to these relations the dorso-lateral and ventro-lateral nuclei are to be regarded as part of the cochlear apparatus. Another nucleus, consisting of specially large cells, lies just ventral to the cerebellum and represents Deiters' nucleus. The remaining nuclei are two in number, one in relation to the descending fibres of the acusticus (the descending vestibular nucleus); the other lying dorso-mesial in the medulla (dorsal-vestibular nucleus). From these fibres pass to the cerebellum and also to enter the fasciculus longitudinalis dorsalis.

Holmes summarises his results by pointing out that the grey matter in which the vestibular nerve fibres end is subdivided into various nuclei, with characteristic structure and connections, which remain nearly constant in the various classes of vertebrates. These nuclei are in all cases closely connected to the cerebellum, to the spinal cord, and probably to the motor cranial nuclei, while in some cases a large commissure connects those of opposite sides.

The fibres from the lagena of the fishes have no end nuclei distinct from those of the rest of the labyrinth; so, then, the cochlea nuclei must be derived from the original tuberculum acusticum of the fishes and are not, as such, represented in the lowest vertebrates. The first indication of specialisation is found in the anura, in which a bundle of fibres homologous to the corpus trapezoides of higher forms springs from the dorsal nucleus, marking this out as a cochlear nucleus. In all groups above the anura a secondary cochlear tract arises from some cells in the acustic area to pass, with more or less interruption, to the region corresponding to the posterior corpora quadrigemina. The size of the cochlear nuclei is directly proportional to that of the peripheral end organ of hearing, variation in this respect being well seen in the reptilia. The cochlear nuclei, unlike the vestibular, have no connection to the cerebellum or spinal cord.

E. HEWAT FRASER.

**PHYSIOLOGY.****THE SEGMENTAL REPRESENTATION IN THE SPINAL CORD  
(220) OF THE SENSORY AND MOTOR FIBRES BELONGING  
TO THE MORE IMPORTANT BRACHIAL NERVES.**

G. BIKELES und M. FRANKE, *Deutsche Ztschr. f. Nervenhe.*,  
Bd. 23, H. 3 and 4.

ON the strength of the results obtained by Cassirer, Flatau, and other observers who have examined the spinal cord for changes produced by the amputation of limbs and by resections of nerves, the authors of this paper determined to apply the latter method for the purpose of defining the central segmental representation of the sensory and motor fibres belonging to the principal peripheral nerves. In order that inferences might be drawn from their experimental results, and applied with some degree of probability to man, they operated on several of the lower animals (*i.e.* rabbits, cats and dogs) for purposes of comparison. In some of these animals they resected the median, ulnar, and musculo-spiral nerves at the same time in the axilla, and in others these nerves were divided separately. The animals were allowed to live four weeks, and the Marchi method was used for the examination of their cords. In a rabbit, a dog, and a cat in which the three nerves were resected in the axilla on one side, many closely aggregated black dots were found in the root zone of the posterior columns of the same side at the level of the 7th and 8th cervical segments and relatively few at the level of the 1st dorsal. In the rabbit the 6th cervical segment showed no entering degeneration, but in the cat and dog that segment contained some degenerated root fibres. Resection of the median nerve alone in the upper arm produced degeneration in the entering root zones of the 7th and 8th cervical segments and to a less extent of the 1st dorsal in all three animals.

Resection of the ulnar showed that most of its afferent fibres entered the cord by the eighth cervical posterior root, and a smaller number by the 1st dorsal, the latter in the rabbit containing less than in the other two animals.

The musculo-spiral was resected at the elbow joint below the origin of the branch supplying the triceps. In the rabbit the majority of degenerated fibres were seen in the posterior root zones of the 7th and 8th cervical segments and a few in that of the 1st dorsal. In the cat there were none in the 1st dorsal, but plenty in the 7th and 8th cervical and a few in the 6th. Unfortunately the material from the dog was spoilt.

Total resection of the brachial plexus above the clavicle was carried out and produced the following results. In the rabbit degenerative changes were found in the entering posterior fibres

from the 5th cervical to the 2nd dorsal segments inclusive—very few in the latter. In the dog the changes were noted in profusion in the 5th, 6th, and 7th cervical segments, but only traces of the same were found in the 1st dorsal segment.

The intramedullary portions of certain anterior root fibres showed analogous changes in their course, and the fibres which showed such changes were traced almost invariably from the region of the lateral groups of cells and very few or none from the median groups.

After complete resection of the musculo-spiral, median and ulnar nerves in the dog, the anterior root fibres were altered in a few instances in the 7th cervical segment, in profusion in the 8th, and in considerable numbers in the 1st dorsal.

After resection of the median, as after that of the ulnar, in the dog, changes were only seen in the anterior root fibres of the 8th cervical segment.

After a high resection of the whole brachial plexus, the fibres of the 6th and 7th cervical segments were affected. In the cat and rabbit changes in the anterior root fibres were not well marked.

In the second part of the paper the authors recount the results of their examination of the ganglion cells by Nissl's method after similar resections, and come to the conclusion that in the dog the motor fibres of the musculo-spiral nerve arise in the 7th, 8th cervical and 1st dorsal segments; of the median in 8th cervical and 1st dorsal; and of the ulnar in the same two segments. As regards the cell groups affected, they found that in all cases in the dog the ventro-median group and the central and anterior parts of the lateral groups were, without exception, free from degeneration. The changes, whether after resection of the median, ulnar, or musculo-spiral (at the elbow), were confined to the dorso-lateral group. When the three nerves were resected together and the triceps branch was included, axonal degeneration was seen in the 7th cervical segment in nearly the whole of the dorso-lateral group, and in the 8th cervical occupied also the so-called "intermediary group" (of Marinesco), so that in the latter segment only the most ventral cells of the lateral groups remained intact. The deduction is that the triceps branch of the musculo-spiral nerve originates in the 7th cervical segment in a part of the dorso-lateral group and in the 8th cervical segment in the intermediary group, a result which agrees with those obtained by Marinesco and his pupils.

The remaining branches of the musculo-spiral originate therefore in the lateral part of the dorso-lateral group, chiefly in the 7th and 8th cervical segments, while the median and ulnar predominate in the median part of this group (this also is in consonance with Marinesco),

In the cat the musculo-spiral nerve appeared to arise from the lower part of the 6th, from the 7th and 8th cervical and from the 1st dorsal segments; the median from the lower half of the 7th, the 8th cervical and the 1st dorsal segments, the ulnar from the same parts as the median.

Corresponding results were obtained in rabbits and guinea-pigs.  
E. FARQUHAR BUZZARD.

**EXPERIMENTAL OBSERVATIONS ON PUPILLARY CONSTRICTION DURING CONVERGENCE AND UPON LATERAL AND CONVERGENT MOVEMENTS OF THE EYES.**

A. MARINA, *Ann. di Nevrol.*, 1902, p. 543.

THE first part of this important paper deals with the constriction of the pupil which occurs during convergence. In a previous paper on the pathology of the ciliary ganglion, Marina wrote as follows:—"What do we really know of the important phenomenon of the survival of pupillary constriction during convergence and on accommodation, after loss of its reaction to light? To call the constriction a 'concomitant' one, does not explain it. Some have imagined the hypothetical pupillary nucleus divided into three parts, one for light, one for convergence and one for accommodation. But Galassi's phenomenon, myosis during contraction of the orbicularis, which may persist when constriction during convergence is lost, would require us to divide the pupillary nucleus not into three but into four parts."

To designate the phenomenon of myosis during convergence a "concomitant" one, means that when the convergence nucleus is stimulated by a voluntary impulse from the cortex, part of the impulse is diverted to the pupillary nucleus, so that the pupil is constricted at the same time as the two internal recti contract. In other words, constriction of the pupil on convergence is supposed to be dependent on innervation of the convergence centre, since the innervation of no other ocular muscle produces pupillary constriction.

In a series of ingenious experiments on monkeys, Marina transplanted the internal rectus into the position of some other ocular muscle, the latter being in turn fixed in the insertion of the internal rectus. The effects on pupillary constriction were then studied. In some experiments, the internal rectus was exchanged with the superior oblique, in others the transposition was between the internal and the external rectus, this latter operation being particularly difficult. In performing it, the two tendons were made to cross, one above and the other beneath the superior rectus, so as not to distort the latter and produce an upward deviation of the eye. In all the experiments the movements

of the eye completely recovered a short time after operation, and subsequent anatomical investigation showed that the tendons were firmly fixed in their new positions.

The result upon the sphincter pupillæ in every case was that constriction occurred when that muscle contracted which had been transplanted into the insertion of the internal rectus so as to produce convergence. Pupillary constriction never occurred when the transplanted internal rectus functionated in its new position. This proves that contraction of the internal rectus, *per se*, has no influence on pupillary constriction, and that the convergence centre has no special bond of union with the supposed pupillary centre. In cases of transposition between the internal and the external rectus, say in the right eye, when after operation the animal directed both eyes to the right, both internal recti were thereby innervated, and yet pupillary constriction did not occur. Therefore the phenomenon cannot be considered as "concomitant" with the innervation of the two internal recti, nor is it dependent on action of the third, fourth, or sixth nuclei.

In another series of experiments, Marina endeavoured to eliminate the influence not only of the central nervous system, but also of the peripheral (ciliary) ganglia. He therefore exposed the internal rectus in a monkey, anæsthetised it with oleo-cocaine (which does not affect the pupil), and stimulated the muscle near its insertion by means of faradism, with the result that every time the internal rectus contracted, the pupil became constricted. Moreover, movement inwards of the eye by means of forceps, producing passive convergence, also caused immediate constriction of the pupil lasting for several seconds and followed by alternate constriction and dilatation. He therefore concludes that pupillary constriction may occur without the action of the nervous system, perhaps from dilatation of the vessels of the iris. But though pupillary constriction may thus occur independent of the central nervous system or of peripheral ganglia, yet Brücke has pointed out that sometimes the pupil of one eye may become constricted when the other eye is directed inwards, thus pointing to a reflex mechanism. Marina suggests an ingenious though hardly convincing hypothesis to account for pupillary constriction not only on bilateral but on unilateral ocular convergence. He points out that the short ciliary nerves lie to the inner side of the optic nerve whilst the long ciliary nerves are external to it, and suggests that the short ciliary nerves are mechanically stimulated whilst the long ciliary branches are stretched during inward movement of the globe. This, however, does not explain the absence of pupillary constriction in the inward-moving eye during the movement of both eyes laterally to one side.

Constriction of the pupil during convergence, then, is a phenomenon which is independent of constriction on accommodation, and the two ought to be clearly distinguished and separately observed clinically. It is well known, for example, that in diphtheritic paralysis constriction on convergence may be present when accommodation is paralysed.

The phenomenon of the Argyll-Robertson pupil, Marina frankly admits, has not yet been satisfactorily explained. He quotes various theories which have been offered to account for it and shows the insufficiency of each.

A further question is as to the existence in the floor of the iter of separate nuclear centres for convergence and for lateral movements of the globes. Marina's experiments show that the movements of convergence can be perfectly performed either by the external rectus or by the superior oblique, if transplanted into the insertion of the internal rectus, and that lateral movements of the eyes can be performed by varied combinations of transposed muscles. It is therefore unnecessary to assume the existence of a special convergence centre or of a centre for lateral movements of the eyes. In other words, *voluntary innervation takes no cognisance either of nuclei or tracts or muscles, but simply of movements and directions.*

The bulbar mechanism of associated ocular movements is still obscure, and Marina holds that in every ocular movement, all the ocular muscles are innervated and that it is the reinforcement of action in a given sense which produces the resulting voluntary movement.

The third, fourth and sixth nuclei were examined in the monkeys after experiment, with the result that after a transient initial change in the affected nuclei, their appearances returned to normal. In one case where the internal rectus was excised, he was able to localise the position in the third nucleus to which it corresponded. Exact details of this and of many other points of interest should be studied in the original.

PURVES STEWART.

## PSYCHOLOGY.

**THE DIRECTION OF INVESTIGATION FOR PSYCHOLOGICAL (222) WORKS.** W. WEYGANDT, *Centralbl. f. Nervenh. u. Psychiat.*, Bd. xxvi., Jan., Feb. u. März, 1903.

THE writer deals with the different mechanical apparatus for estimating the time required by different individuals for various mental acts. He includes tests for memory, for reading, writing, counting numbers and figures, and especially studies the onset of fatigue in each of these exercises.



The apparatus devised by Finzi is described, which, by means of a mechanism, allows cards bearing letters or numbers to be exposed for a known length of time—0·167 seconds. Observations as to the length of time it takes different subjects to recognise numbers or letters or groups of such are made. On an exposure being made the subject at once says or writes down as many figures as he remembers, and places them in the same order as that in which they were exposed. The latter part of the experiment was the more difficult. Some individuals were found to recognise certain letters more quickly than others. It was found that the number of letters named correctly increased directly as the length of time after exposure up to thirty seconds. After a longer interval the faculty for remembering is not further improved, but rather deteriorates. A certain length of time is required for an image to become sufficiently imprinted on the memory as to be reproduced. The subjective feeling of certainty depends upon the activity of the memory in building up impressions; it is greatest soon after the act of comprehension, later it decreases, and is influenced by doubt which frequently gives rise to false impressions.

Through exercise the certainty of comprehension and memory increases.

Kraepelin and Goldscheider's instruments for recording the results of writing experiments are referred to and described at considerable length.

Griesbach's observations with the aesthesiometer with Bolton's modification of the instrument are noted. The researches of Axel Öhrn, and their elaborations by Cattell, Berger and Ebbinghaus are next treated of at great length. These investigations principally treat of the onset of fatigue during various mental work, *e.g.*, counting, repeating and adding numbers or letters, and writing to dictation.

The method was to count letters continuously, the time being taken at regular intervals, so that it could be seen whether there was any alteration in the number counted in each period as the subject became fatigued. By taking many observations a curve of the numbers was formed. In this way the onset of fatigue, as evidenced by a lowering of the curve, was noted. A similar principle was followed in writing to dictation and in the addition of numbers. Ten people were made the subjects of experiment; care was taken to eliminate error as far as possible by conducting the observations day by day under similar conditions.

In letter-counting there are two principal acts, *viz.* (1) the perception and recognition; (2) the utterance. The perception and recognition occupy most of the time; the utterance, in educated people, is done so quickly as to be more or less reflex.

The time is spent in the central act in arousing attention and giving rise to recognition.

Fatigue therefore follows from the first part. To comprehend and pronounce a number occupies, on an average, 0.406 seconds. The average times for the acts of group naming, addition, writing to dictation, reading, number and syllable learning are .323, .1244, .435, .138 and 9.6 seconds respectively. With regard to addition, great individual differences prevail. The rule laid down is that the length of time taken is a criterion of the complexity of the act.

Exercise causes a delay in the onset of fatigue. Usually at the commencement of the experiment the time is lengthened, this is ascribed to "adaption time." Here the curve sinks, but it is only transient, and almost immediately the curve rises and continues at a uniform height until the onset of fatigue, then it falls.

The researches of Brettman and Miesmer are mentioned, in which they sought to estimate the difference between mental and bodily work in their effect on reaction time, and to show its marked lengthening after mental effort. The paper concludes with an analysis of the curve of a mental act, and the conclusions closely coincide with those of Kraepelin, who showed that in conditions of mental vigour, energy is abundant, and mistakes are few, but that gradually as the day goes on, fatigue sets in and is evidenced by mistakes gradually increasing in number, by delayed reaction and diminished attention.

W. MAULE SMITH.

**THE FACULTY OF ORIENTATION FROM A DISTANCE.** By ED. (223) CLAPARÈDE, *Arch. de Phys.*, March 1903, p. 134.

THIS article is to a large extent a review of recent literature on the subject of orientation. The author discusses very briefly the numerous theories which have been suggested to account for the facts of orientation from a distance. For any full consideration of these theories the writer does not think we are ready. Many of the facts they are intended to explain are themselves open to doubt, or at any rate require more careful study before we can even state clearly the problems which await solution. Accordingly the greater part of the paper is devoted to the consideration of the facts at present known. Facts concerning the migration of birds, the homing instinct of carrier pigeons, the return of cats and dogs to the house from which they had been removed, the sense of direction in ants and bees, are cited and arranged according to their bearing upon various questions, such as the route taken upon a return journey, the influence of the sun, of winds, and atmospheric conditions upon the sense of direction and so on. Although

the literature bearing upon such questions is very copious, the writer believes that very many of the observations and experiments which have been made require to be repeated under more stringent conditions, and that all efforts should for the present be directed to this end rather than to the discussion of theories.

W. B. DRUMMOND.

### CLINICAL NEUROLOGY.

**ON THE PATHOLOGY AND PATHOLOGICAL ANATOMY OF**  
(224) **TOXIC POLYNEURITIS AFTER THE USE OF SULPHONAL.** W. ERBSLÖH, *Deutsche Ztschr. f. Nervenhe.*, Bd. 23, H. 3 u. 4.

AMONG the various forms of toxic polyneuritis, the one caused by alcohol has been best studied both clinically and pathologically; those due to arsenic, mercury, carbon dioxide, and carbon bisulphide have received a considerable amount of attention; while that which owes its origin to the employment of sulphonal as a drug has hitherto not been investigated from the standpoint of morbid anatomy. Dr Erbslöh records briefly Wien's case of sulphonal poisoning which ended fatally, but was not examined microscopically, and then proceeds to relate the clinical symptoms and pathological findings in a fatal case of his own.

A woman, forty years of age, who had previously been perfectly sane and of temperate habits, was admitted into hospital in order to undergo the operation of hysterectomy for carcinoma of the uterus. Owing to sleeplessness about the time of the operation, she was given 2 grammes of sulphonal every evening for five days; five days after the last dose she first complained of pains in the legs. Muscular paresis followed and spread rapidly, so that ten days later her condition was briefly as follows:—T. normal. Pulse 80-100. Thoracic and abdominal organs apparently healthy. Urine contained hæmatoporphyrin. Right pupil larger than left, both reacted normally. Slight left external rectus palsy. Paresis of both legs and arms, affecting the proximal more than the peripheral muscles. Neck and abdominal muscles also paretic. No R.D. Blunting of tactile and painful sensibility on legs and trunk. Sense of position and thermæsthesia intact. Knee-jerks absent. Plantar reflexes normal. Intelligence, memory, and knowledge of time and place good. Hallucinations, especially at night.

A week later the paralysis was more marked, and there was partial reaction of degeneration in the muscles. Deep and superficial reflexes were absent. Death ensued through respiratory failure.

*Post-mortem.*—There were no macroscopical changes of importance. By the Nissl stain the anterior horn cells of the upper

cervical region were apparently normal, and the nerves of the brachial plexus showed no changes by the Marchi method. The lower part of the cord does not seem to have been stained by Nissl, but the anterior crural nerve showed extensive degenerative changes in the myelin sheaths by the Marchi method, and swollen and irregular axis cylinders by Kaplan's stain. This condition was more marked in the peripheral parts of the nerve. In the connective tissue lymph spaces of this nerve the author describes the presence of numerous fibrin threads, numerous lymphocytes, but very few leucocytes. The deltoid and quadriceps muscles showed no change by Marchi.

The author concludes that the morbid process began in the peripheral parts of the nerves and spread centralwards, and that it consisted primarily of a parenchymatous degeneration of the myelin sheaths and axis cylinders, to which was added secondarily a certain amount of inflammatory reaction.

E. FARQUHAR BUZZARD.

**TUBERCULAR POLYNEURITIS WITH EXAGGERATION OF  
(225) THE TENDON REFLEXES IN THE RABBIT.** DE BUCK,  
*Journ. de Neurol.*, March 20, 1903, p. 143.

THE author in this paper endeavours to explain the exaggeration of the tendon reflexes not infrequently met with in peripheral neuritis. The statements made by Strümpell and Moebius, Brissaud and Bruandet, Babinski, Dejerine, Crocq and others are quoted in considerable detail, all admitting this clinical observation as an undoubted fact and offering various explanations.

Dr de Buck by means of an experiment offers a pathological basis which he considers sufficient to explain the phenomenon. A rabbit was inoculated intravenously with tuberculosis. For two months it had been increasingly paralysed, and at the time of death all movements in the lower limbs were abolished. The tonicity of the muscles was greatly increased and there was great exaggeration of the tendon reflexes in the lower limbs. The sphincters were healthy.

Microscopically the muscles of the lower limbs were but little atrophied and there was no degeneration of muscle fibres or any evidence of myositis. The nerves of the lower, and to a less extent of the upper limbs, presented the characteristics of a parenchymatous degeneration, the axis cylinders swollen, vacuolated in fragments and in part absorbed, and the myelin sheaths were atrophied; but in no part was there the typical fragmentation of myelin found in Wallerian degeneration. At a more advanced stage of degeneration shapeless granular protoplasmic masses were found, with, here and there, the proliferated nuclei of the neurilemma

sheaths. The cells of the anterior cornua in the sacral and lumbar regions of the cord and of the posterior nerve root ganglia were healthy and showed no change either in the position of their nuclei or the arrangement of the chromatic granules. The anterior and posterior roots were little if at all affected, but the antero-lateral tracts showed diffuse changes both in the axis-cylinders and myelin sheaths similar to those found in the peripheral nerves. Dr de Buck believes that this is due to a toxic parenchymatous neuritis limited to the nerves and antero-lateral tracts of the cord, but without the cells being affected either primarily or secondarily.

The exaggeration of the tendon reflexes may be due therefore to: (1) irritation of the peripheral nerves, the cord being healthy; but it may also be due to (2) the cord lesion referred to above cutting off the inhibitory influences of higher centres and thus permitting the excessive, because unrestrained, irritability of the tendon reflex centres.

The methods used were formaline as a fixative and van Gieson's stain for the nerves, and Flemming's solution for the muscles to demonstrate the absence of any degeneration of the muscle fibres. The paper is based on the examination of practically one rabbit, and although it is certain that a toxin may act more powerfully on a cell process than on the cell centre, more evidence is desirable before the statements made can be considered beyond the realm of conjecture.

ROBERT A. FLEMING.

**A CASE OF MULTIPLE FIBROMATA CONFINED TO THE**  
(226) **INTERNAL PLANTAR NERVE.** W. J. TAYLOR and W. G.  
SPILLER, *The Journal of Nervous and Mental Disease*, April 1903,  
p. 204.

THE patient, a young woman of 27, was first seen in 1888. Her family history was good. When 16 she had sprained her left ankle, which had always been weak since. Her previous health had been good. When 20 she began to suffer from pain in the sole of the left foot and inner side of the ankle; at first the pain was only present on pressure but gradually it became worse and was only absent when the patient was lying down. For two years previous to her being first seen her work necessitated a great deal of standing and this aggravated the condition, which became more localised and was confined to spots along the inner side of the foot and ball of the great toe. After acute attacks of pain the foot would swell and become red, also if she sat with the foot in a dependent position. On examination small masses were felt along the course of the internal plantar nerve which were hard, movable, and exquisitely tender. Some of them seemed to be immediately

beneath the skin. Profuse sweating occurred over the whole foot, especially on the sole.

She was operated on in 1888, 1890, 1897, with temporary relief, nineteen neuro-fibromata being removed. Ultimately in June 1902, five inches of the swollen nerve were removed by Dr Taylor and patient had complete relief. Dr Spiller who examined the tumours first cites Recklinhausen as to the rarity of neuro-fibromata in the hand and sole of the foot. The vessels removed with the tumour were diseased with thickened walls and in some cases the lumen was obliterated. The author suggests that the growth started in the endoneurium, and that though many nerve fibres were degenerated yet fresh ones were formed, enabling the painful stimuli to be transmitted. Dr Spiller points out that in this case the long duration and repeated operations which are stated by some to be liable to cause sarcomatous change did not have any such result in this case. T. GRAINGER STEWART.

**ON LESIONS OF THE CAUDA EQUINA AND ON RADIOLAR**  
(227) **INNERVATION.** PANSINI, *Rif. Med.*, Jan. 7, 14, 21, 28, and  
Feb. 4, 1903.

HAVING met with six cases of lesion of the cauda equina during the past year the author has been able to make an elaborate clinical investigation of the functions of this portion of the spinal cord. The roots which form the cauda equina are so arranged that the pairs which arise from the lower segments of the cord lie internal to those which spring from the upper segments, and as each pair of roots, forming a root segment, is connected with a medullary segment, it is evident that the same series of symptoms will follow a lesion of the one as of the other.

The region of the cord from which these roots arise may be divided into three sub-regions, the cone, the epicone, and the upper half of the lumbar enlargement; and these sub-regions correspond peripherally with the pudendal, sacral, and lumbar plexuses.

The upper limit of the cone is usually placed by anatomists at the level of the origin of the 5th sacral root; but Raymond, on clinical grounds, places its upper limit at the level of the 3rd sacral root, and this view is supported by clinical observations and by some anatomico-histological facts regarding the changes in the arrangement of the nerve fibres and nerve cells of the cord which take place at the level of the third sacral root.

The term epicone is applied to the portion of the spinal cord, which extends from the cone to the 5th lumbar segment, from which the roots of the sacral plexus arise. The upper limit of the epicone cuts the lumbar enlargement almost in half, and the upper half of the lumbar enlargement forms the third division of

this region. Corresponding with these three divisions, three morbid types in the pathology of the cauda equina and of the lumbo-sacro-coccygeal cord are described, and each of these types may be again divided into two sub-types according to the partial or complete injury of the portion affected.

In Case I. there was a small lesion of the grey substance of the cone and of the lower part of the right anterior cornu of the epicone.

The most marked symptoms were paralysis of the bladder and generally obstinate constipation, but incontinence if the fæces were at all liquid after the use of a purgative. Objective sensation was normal generally, the knee-jerk was increased on the right side, the cutaneous reflexes were intact. There was also slight wasting of the muscles of the calf and of the peronei on the right side with fibrillary contractions. The lesion here was of syphilitic origin and was limited to the centres of micturition and defæcation, and to the grey matter of the epicone up to the 2nd sacral root. The absence of any anæsthesia of the perineum, penis, scrotum, anus, and sacrogluteal saddle, show that only a very minute portion of the cone was affected.

The syphilitic lesion in these cases consists of a small celled infiltration or of small gummata, which affect the veins more than the arteries and cause thrombosis and softening.

The next four cases were all caused by falls on the nates, and the sixth by a bullet wound.

In Case II. there was weakness of the lower limbs, retention of urine requiring catheterisation for one month, and after that incontinence and spontaneous passing of fæces; erections absent. There was also anæsthesia of the penis, scrotum, perineum, upper internal portion of thigh, and sacro-gluteal saddle, and of a zone on the posterior surface of the thighs extending downwards between the semimembranosus and the biceps. On the outer side of the feet there was a narrow band of diminished sensation. The anus and urethra were also anæsthetic. The anæsthesia to pain and cold was complete and almost complete to touch. There was slight hyperæsthesia of the borders of this zone. The muscles of the leg and back of thigh were flaccid, but there was no true paralysis; fibrillary twitchings were present. The anal reflex and Achillis jerk were lost; the cremasteric and abdominal reflexes were preserved; knee-jerk increased; testicular pain present. This ano-urogenital disturbance of function and sensation, combined with anæsthesia of the perineum and sacro-gluteal saddles, point to a lesion of the cone, and there were no symptoms to suggest any lesion of the roots. The region of the sacrogluteal saddle is connected with the third and fourth sacral roots, and the area between the semimembranosus and biceps with the second sacral

root (Thorburn); the external border of the foot must also be connected with this second sacral root.

The lesion which caused the motor disturbances extended to the 2nd sacral segment. The fact that in lesions of the cone the motor disturbance is more extensive than the sensory has been noticed by many observers.

We have here, therefore, a case of lesion of the cone (including the 3rd sacral segment), and a slight disturbance of the epicone.

Case III. This patient had shooting pains in the nates and legs after the fall; he had also retention of urine and involuntary loss of fæces.

The area of anæsthesia spread beyond that seen in the last case and affected the popliteal space and the back of the leg to the intermalleolar line; also the whole of the foot outside the line of the first interdigital space.

The mobility of the toes and feet was reduced; flexion of the knee and external rotation of the thigh were weak. Fibrillary contractions were seen in the gluteal, calf, and peroneal muscles, and to a lesser degree in the posterior muscles of the thigh.

The plantar and anal reflexes and the Achillis jerk were abolished; the cremasteric and abdominal reflexes were present, and the knee-jerk was increased. The erections were weak and ejaculation was by drops.

In this case the cone and part of the epicone were injured; the 2nd sacral root is certainly wounded, and probably the 1st sacral. The area which corresponds to the 1st sacral root, *i.e.*, the root immediately above those affected in the last case, includes the posterior and outer side of the leg and the intermediate portion of the foot. Moreover, we find from this case that the internal side of the foot is innervated by a higher root than the outer. This agrees with the scheme of innervation drawn up by Kocher, but is opposed to that of Thorburn. The motor disturbance spread to the 1st lumbar root, and we thus find that in this case the cone and part of the epicone were affected.

Case IV. Immediately following the fall this patient had violent pain in the sacral region and his feet felt dead. There was difficulty in micturition, with incontinence after about twelve days, and there was early constipation, but incontinence after the administration of a purgative. Erections were absent but they reappeared after a time. In this case the whole of the foot excepting a small triangular area in front of the internal malleolus was anæsthetic, and the anæsthesia also spread on to the antero-external side of the leg. Inflammation followed the pricks made in mapping out the areas of anæsthesia.

The movements of extension of the knee were normal, but



flexion was weak and external rotation of the thigh impossible; cramps were frequent. The anal and plantar reflexes, and the Achillis jerk and knee-jerk were absent, the gluteal, cremasteric and abdominal reflexes, were intact.

The anæsthesia in this case exceeded that seen in the last case in that the anterior part of the inner side of the foot and a larger part of the anterior and external surface of the leg showed loss of sensation. If therefore the outer border of the foot is innervated by the 2nd sacral root, and the intermediate portion by the 1st sacral root, this added area must be supplied by the 5th lumbar root. This differs from Kocher's statement that the whole foot is innervated by the 1st and 2nd sacral.

The motor disturbance extended upwards to the 4th lumbar root; this was proved by the weakened action of the quadriceps to electrical stimuli and by the loss of the knee-jerk.

We have therefore an affection of the whole of the sacral plexus (cone and epicone), and from an examination of this case we see that the sensory and motor innervation of the plexus coincides with that of the roots of which it is composed.

Case V. The fall in this case was followed by loss of sensation and of motion in the lower limbs, with pain over the sacrum, and attacks of lancinating pains and cramps in the limbs; at the same time there were priapism, retention of urine and obstinate constipation for a fortnight; afterwards there was some control over the bladder and rectum.

The anæsthesia was complete over the area supplied by the epicone, less marked over the area supplied by the cone, and only slight over the area supplied by the remaining portion of the lumbar enlargement, *i.e.* the upper and outer part of the thighs and the pelvis, excepting those parts supplied by the sacral roots mentioned in one of the earlier cases. In this case also pustules often followed the pricks of a needle. The feet were immobile, flexion and extension of the knee were limited and weak; movements of the thigh were weak. Fibrillary contractions were seen in the muscles of the nates, thighs and legs. Plantar reflex, Achillis jerk and knee-jerk were lost, anal reflex slight, cremasteric reflex intact. Erections frequent but incomplete, ejaculations irregular, coitus possible but accomplished with difficulty.

In this case all the lumbo-sacral segments were affected to a greater or less degree.

Case VI. This patient ten years ago was hit by a bullet at the junction of the last lumbar vertebra with the sacrum, which produced a condition of anæsthesia exactly similar to that found in Case IV., excepting that here the lesion was entirely limited to the right side; and it must have been a lesion of the roots because the injury was much below the level at which the medullary cone

ends (2 lumbar vertebra). Micturition was regular and he had obstinate constipation for a short time only. Erections were incomplete, ejaculations normal.

From this case we learn that with lesion of the roots on one side the functions of micturition, defæcation, and ejaculation, are little disturbed, that erection is incomplete because the corpus cavernosum on the same side does not become turgid, that muscular atrophy with fibrillary contractions follow a lesion of the roots, that there is a hyperæsthetic border to the areas of anæsthesia with lesion of the roots and that the reflexes are increased on the opposite side of the body.

In these cases we have seen examples of all the clinical forms of lesion of the cauda equina; in the first case there was a lesion of a small portion of the cone with an extension upwards into the grey matter of the epicone on the right side, due to a syphilitic affection of the vessels; the next four cases showed lesions of (*a*) the cone, (*b*) the cone and part of the epicone, (*c*) the cone and the epicone, (*d*) the cone and all the lumbar enlargement. Case II. was certainly due to a central lesion. In discussing the question of the seat of the lesion in Cases III., IV., V., the author mentions that the great majority of lesions of the cone are of traumatic origin, and that fracture of a vertebra is often present, which can be recognised by the fixed pain and by the tenderness on pressure over the seat of injury. But the trauma may also give rise to a hæmatomyelia or an acute transverse myelitis without fracture, and these two conditions may exist together, or either may be present without the other; and, moreover, an inflammation of the roots may accompany a traumatic myelitis (Raymond and Cestan).

The most important symptoms which suggest a lesion of the roots are: (1) Pain in the regions of the paralysed muscles. It is true that pain may be present in cord lesions if the posterior columns are affected, but not if the lesion is limited to the grey matter; in these cases, however, the pain is not so intense or so persistent;

(2) The course of the illness. In lesions of the cord the cases usually get steadily worse, while with lesions of the roots there is generally some improvement.

The value of these two symptoms is not absolute; notice should also be taken of (1) a syringomyelic disturbance of sensation, (2) fibrillary contractions, (3) trophic disturbances, (4) symmetry of the paralytic symptoms. These, although they are not of much value taken singly, are of some assistance when associated with the others mentioned.

Another important means of diagnosis is lumbo-puncture. The presence of blood in the cerebro-spinal fluid withdrawn is evidence of injury of the cauda, but it does not exclude a

simultaneous lesion of the medulla. In Cases IV. and V. lumbo-puncture gave negative results.

The examination of the fluid withdrawn will also enable us to distinguish between acute and chronic affections.

The conclusion is arrived at by the author that in Cases III., IV., V., the paraplegia with pain suggests a lesion of the roots, but the course of the disease is in favour of a lesion of the medulla as also are the fibrillary contractions, and the symmetry of the symptoms. Moreover, lumbo-puncture in Cases IV. and V. demonstrated the integrity of the meninges.

Finally, he gives the diagnosis of traumatic myelitis in cases II., III., IV. and V.

#### *Considerations of Localisation.*

While recognising the insufficiency of such a small number of clinical cases to determine the zones of sensation, the author suggests the following scheme as the order of these zones :—

1. The sacrococcygeal region is the lowest.
2. Then follow the perineal and scrotal regions with the sacrogluteal saddle, and a zone on the back of the thigh between the semimembranosus and biceps.
3. External third of foot.
4. Posterior face of leg and posterior part of the peroneal face of the leg.
5. Intermediate third of foot.
6. Anterior zone of peroneal surface of leg.
7. Internal face of leg.

These correspond with the scheme of Kocher and demonstrate that the external surface of the leg and foot is innervated by roots of lower level than the inner side. But Kocher connects the whole foot with the 1st and 2nd sacral roots, while the author adds the 5 lumbar on the inner side.

For several reasons it is unsafe to judge absolutely of the root innervation of the muscles from the innervation of the skin over them. Kocher has shown that with fracture of a vertebra the motor paralysis is always much more extensive than the disturbance of sensation. This is also seen in Pott's disease, in cases of myelitis from compression, and is supported by the cases described in this article. The author suggests that it may be due to the relatively greater vulnerability of the motor-fibres. He submits the following table, however, as the order of root innervation of the muscles of the leg from below upwards, muscles of feet, peronei and extensors of feet and toes, glutei, tibials, and flexors of the feet, flexors of knee, quadriceps, adductors.

The author then proceeds to discuss the position of the centres of the rectum and the urogenital organs. Anal and urogenital

disturbances constitute the fundamental symptoms of lesions of the cauda equina and to these sensory and motor symptoms may be added. Budge localised the centre for the bladder at the level of the 4th sacral segment. Gianuzzi places one centre at the level of the 3rd sacral whose action is conveyed by the sympathetic, and another at the 5th sacral level, whose action is conveyed through the hypogastric plexus. There must certainly be one centre for the detrusor and another for the sphincter which are antagonistic to each other; but it is necessary to admit a third centre for the sphincter of smooth muscle, which is innervated by the sympathetic system. In some of the cases mentioned in this paper in which with a lesion of the cone there was paralysis of the voluntary sphincter, while the sphincter of smooth fibres, which is innervated by the sympathetic, remained intact, there was obstinate retention of urine for about fourteen days, followed then by a condition of what is termed "imperious micturition at short intervals," in which the bladder is never completely emptied, but expels small quantities frequently. No true incontinence or continuous dripping was present.

On account of the varieties of bladder disturbance met with in these cases and in cases of acute total transverse myelitis of other parts of the cord, the author suggests that the correlation of these centres with the higher parts of the cord and with the brain must be very complicated. Czyhlarz and Marburg, from clinical studies in relation with cerebral lesions met with after the autopsy, suggest a double centre of micturition in the brain, one in the caudate nucleus and the other in the putamen of the lenticular nucleus.

The pathological variations of defæcation are analogous to those of micturition

Case VI. with a one-sided lesion confirms the view that the spinal centres for micturition and defæcation are equal on the two sides.

With regard to the genital functions the author states that the centres for erection and ejaculation are at a higher level than those of the bladder and rectum, and he locates the centre for ejaculation in the 3rd sacral segment and that for erection in the 2nd sacral. It is necessary to admit a sacral centre for erection because in all lesions of the cone erection is incomplete. From this it will be seen that one of these functions may be interfered with without the other.

Case V., in which there was a priapism for fourteen days, suggests that there is a second centre for erection at the junction of the last dorsal and first lumbar segments, in which case the priapism may have been due to a hyperexcitability of a centre placed between the healthy and diseased parts.

Up to this point the author has dealt only with the clinical evidence of zones of sensation. He now proceeds to discuss the bearing of morphology and embryology on the limitation of these zones. He insists on the importance of observing the law of Sherrington, that each region of the skin is innervated by at least three roots, and the law of Sherrington, Thorburn, Dejerine and others, that each muscle is innervated by at least two roots. He then refers to the development of the various parts of the body from a number of segments or metameres placed in series, and mentions that the upper and lower limbs are each developed from the ventro-lateral part of two series of these metameres.

The upper series from which the upper limb is developed consists of the 4th, 5th, 6th, 7th, 8th, 9th (1 D), and 10th (2 D) metameres. From the antero-lateral part of these there is a growth outwards to form the limb. The 4th and 10th end at the shoulder, the 4th at the acromial process, and the 10th at the axillary fold. The arm itself is thus formed from the 5th, 6th, 7th, 8th and 9th metameres.

If we consider the arm as a growth at right angles to the trunk with the radial border uppermost, we shall find that these five metameres lie in the order mentioned above, and that the 5th is on the radial or upper side. The 5th and the 9th do not extend beyond the wrist; the 6th, 7th and 8th go on to form the hand, the 6th the thumb, the 7th the intermediate portion of the hand, and the 8th the little finger.

Applying the law of Sherrington to this scheme we find that the thumb, which corresponds to the 6th metamere, is innervated by the 6th root and also by the 5th and 7th roots; the intermediate portion of the hand by the 7th root and also by the 6th and 8th; the little finger by the 8th root and also by the 7th and 9th. The law of development then explains completely the sensory innervation of the upper limb, and shows that it is exactly metameric. The same law applies approximately to the lower limb. Seven metameres take part in its development also, the 2nd, 3rd, 4th and 5th lumbar, and the 1st, 2nd and 3rd sacral. The 2nd lumbar and the 3rd sacral end at the hip joint, and the leg is developed from the 3rd, 4th and 5th lumbar and the 1st and 2nd sacral.

Morphology shows that of these the 3rd lumbar and 2nd sacral extend to the ankle, and the foot is innervated from within outwards by the 4th and 5th lumbar and the 1st sacral.

The results obtained by the author from his clinical cases differ from these somewhat, but they uphold the view that the outer side of the foot is innervated by lower roots than the inner side. The author has found clinically that the area of the 3rd lumbar root does not extend as far as the ankle, but that this with a small triangle in front of the internal malleolus is innervated by the 4th lumbar.

He connects the great toe with the 5th lumbar, the intermediate portion of the foot with the 1st sacral, and the outer side of the foot with the 2nd sacral.

Morphology also proves that the external genitals are innervated by lower roots than those which innervate the lower limbs.

According to Ostroumoff the penis and all the external genitals are the equivalent of a third pair of limbs.

After referring to the areas which correspond to the plexuses and the peripheral nerves the author offers the following conclusions on the sensory supply of the limbs.

(1) The distribution of the plexuses corresponds almost exactly with the root distribution.

(2) The peripheral terminal distribution coincides in many points with that of the roots, and is at any rate fundamentally metameric.

(3) The law of Sherrington establishes that every sensory region is innervated by three roots; on the other hand if a nerve is cut the area is anæsthetic. The overlapping must take place below the roots and above the peripheral nerve, that is in the plexuses.

Their function then is to secure the multiple innervation of every peripheral point.

But if a sensory nerve is cut, the periphery is not absolutely insensible; there exists a recurrent sensibility which is due to terminal anastomosis; and the intercostal nerves form no root plexuses and still the law of Sherrington holds good; in these also there must be terminal plexuses.

The attempt to attach to every root a definite motor function has not been very successful. It is probable that the motor innervation has a metameric basis; every muscle, also, is innervated by at least two roots.

The author then gives a long table of the muscles with their root innervation and the sensory root innervation of the overlying skin. This shows that there exists an undeniable relation between the two.

To this table there are exceptions, but these are, many of them, easily explained, *e.g.*, the large fan-shaped muscles of the trunk, pectoral, trapezius, etc., are innervated according to their movable attachment and not to their fixed point.

Others are much more difficult to explain.

With regard to the arrangement of the fibres of deep and muscular sensibility not much is known, but it is probable that they follow the same laws as the motor and sensory fibres, and have a metameric distribution.

After section of the roots and peripheral nerves we find the motor results analogous to those seen in section of the sensory

nerve. Section of one root does not produce complete paralysis, section of the nerve below the plexus does. The intrinsication must therefore take place in the plexuses. R. G. Rows.

**TABES IN ASYLUM AND HOSPITAL PRACTICE.** By F. W. MOTT, (228) M.D., F.R.S., *Archives of Neurology*, vol. ii., 1903, pp. 1-327.

THE author of this valuable paper has had exceptional opportunities of observing diseases both in hospital and asylum practice, and he has studied all types of tabes from cases presenting purely spinal symptoms to cases presenting mental symptoms which would lead to a diagnosis of general paralysis. The paper is based on notes of more than sixty cases of tabes and sixty cases of the tabetic form of general paralysis. He has obtained thirty post-mortem examinations and made a systematic examination of the nervous system of each. Owing to the vast amount of material, he has limited his observations to one class of disease, viz., tabes dorsalis, thinking it possible "to associate some of the mental disturbances with the changes which one meets in the nervous system, and to show that the illusions, delusions and hallucinations have an organic basis, and that there is a great analogy between the paroxysms of pain, the visceral crises of tabes, and the epileptiform fits, attacks of mania, delirium, hallucinations and other mental disturbances which occur in general paralysis."

Notes are given of the current views of the relation of tabes to general paralysis. His own view is that "pathogenetically, the two diseases are identical"; that "etiologically and pathogenetically there is *one tabes* which may begin in the brain (especially in certain regions), or in the spinal cord in certain regions, or in the peripheral nervous system connected with vision, or in nervous structures connected with the viscera, constituting, therefore, different types, any of which may be present or be associated with one or all of the others." These different types, too, of a single pathogenetic morbid entity have "a close clinical and pathological relationship." He strongly opposes the view that the appearance of cerebral symptoms in a tabetic case is due to propagation by direct continuity of diseased tissue—it is *not* an ascending change.

*The Etiology of Tabes and Taboparalysis.*—He thinks that we are coming more and more to the opinion that syphilis is the cause of the degenerative process, or at least the main causal factor; the fact that we cannot prove more than 70 or 80 per cent. of patients to have suffered from syphilis is no argument against this doctrine, and he thinks it possible that some of the cases of tabes or general paralysis occurring in adults, in which no history of acquired syphilis can be obtained, may owe the disease to an inherited taint; in two of his cases of tabes there was a history of congenital

syphilis. His own statistics, as regards syphilitic antecedents, are as follows: Of 47 male tabetics, 33 (over 70 per cent.) had certainly syphilis, and only one of the others could say that he had not been in the way of getting it. Of 54 male cases of taboparalysis, at least 75 per cent. had syphilis, and in only one case was there absence of history and of reliable signs. Of 8 female cases, it was "almost certain" that all had been infected. A comparative analysis of the birth-rate of the two classes of cases shows that the taboparalytics have fewer children, healthy and dead; Mott's statistics also show very markedly that, when the female is infected, the chance of living children being born is greatly reduced. Nearly 30 per cent. of the married women were sterile. (This agrees very closely with Mendel's results.) There were seven examples of affection of married couples; in all cases it was probable that the husband infected the wife, and signs of syphilis were observed or indicated in either husband or wife in every case, with the doubtful exception of one. This conjugal affection is taken as an important argument in favour of the syphilitic origin of the disease, while the fact that one of the pair may be affected with tabes and the other with paralysis supports the view of the unity of the two diseases. Of 32 cases of the *juvenile* form of general paralysis, at least 80 per cent. suffered from congenital syphilis, and probably every case was of syphilitic origin. The syphilitic view is also strengthened by the results of post-mortem examinations. In 213 male post-mortems there were 86 general paralytics, and 45.4 per cent. of these had signs of syphilis on the body; while of the remaining 127, 9 per cent. had certain or doubtful signs of syphilis. In 249 female post-mortems there were 36 general paralytics, and 19.5 per cent. of these had signs of syphilis; while of the remaining 213, less than 2 per cent. showed signs of syphilis. Mott reconciles the two contradictory facts, that races free from syphilis are free from tabes and paralysis, and that certain races extensively syphilised do not suffer from these two diseases, by supposing that other "contributory factors" are absent in the latter. Among these factors are enumerated: (1) stress; he quotes instances in which the stress of occupation has apparently determined the seat of lesion: thus, among 15 female tabetics, most of whom had much kneeling to do, there were seven cases of arthropathy, and in 6 the knee was affected, while only 5 of 54 male tabetics suffered from joint affections; (2) mental worry and strain; (3) exposure to cold and wet; (4) injury to the spine; (5) hereditary predisposition—not so important in tabes as in general paralysis; among the taboparalytics and insane tabic cases, 30 per cent. had a family history of insanity; (6) intemperance, more important in taboparalysis than in tabes, not so much in the production of the disease as in accelerating its progress.



*Modes of Onset.*—Of 65 cases of tabes, the average age of onset was 37, the youngest 25, the oldest 55; average interval between infection and onset of symptoms was 15 years, shortest 4, longest 26 years. Of 54 taboparalytic cases, the average age of onset was  $38\frac{1}{2}$  years; average interval after infection was 15 years, shortest 6, longest 25 years. In 34 cases, the average age of onset of spinal symptoms was  $37\frac{1}{2}$  years, of mental symptoms  $39\frac{1}{2}$  years; in one-half of the cases the spinal and mental symptoms were apparently simultaneous in onset; in 24 of 48 cases, signs of brain affection either preceded or were simultaneous with signs of affection of the cord or eyes or both.

#### SYMPTOMATOLOGY.

*The Eye Symptoms.*—The hospital cases of tabes closely correspond to the asylum cases as regards pupil phenomena: inequality of the pupils is more frequent in the asylum cases, paralysis of the ocular muscles much more frequent in hospital cases.

*Optic Atrophy.*—Of 65 hospital cases, 20 had optic atrophy; while, of 60 asylum cases, 35 per cent. had well-marked optic atrophy, and probably 50 per cent. would be nearer the mark. In Mott's opinion, optic atrophy occurring in tabes is a serious indication of the possibility of the degenerative process attacking the brain. He opposes Benedikt's law that the tabetic motor symptoms, no matter what development they may have reached, vanish as soon as optic atrophy appears: he believes that optic atrophy—like signs of cortical degeneration—has no influence on the ataxy when it has reached the second stage, although it may arrest the spinal degenerative process and modify ataxy of the first degree.

*Sensory Disturbances* are dealt with at considerable length. Of 48 successive cases of tabes, 42 showed objective cutaneous sensory disturbances; the remaining 6 were all in the preataxic stage. Trunk anæsthesia to light tactile impressions, usually about the fourth or fifth dorsal areas, is the earliest and most constant sensory disturbance (present in 36 cases). Analgesia or hypalgesia may also be present, but usually over a less extensive area; hyperæsthesia or hyperalgesia may precede anæsthesia or analgesia, and often there is a hyperæsthetic area above or below the anæsthetic region or a patch of hyperæsthesia may be situated within an anæsthetic area; in the hyperæsthetic area, there was generally hypersensitiveness to cold. In 33 cases, there was sensory affection of the legs, mainly analgesia or hypalgesia below the knees, often also light tactile anæsthesia, frequently associated with anæsthesia of the genital, perineal and anal regions. In 12 cases, the arms were affected by extension upwards of the thoracic anæsthesia; in only 3 cases did the anæsthesia occur on the radial side of the

median line. Among taboparalytics, analgesia was fairly common ; loss of the painful sense, with retention of tactile, was more often met with than in tabes ; anæsthesia and analgesia were found in at least 42 per cent. of the asylum cases ; the appearance of the mental symptoms in some cases undoubtedly diminished not only the ataxy but also the anæsthesia and analgesia. Two diagrams are given showing the distribution of the anæsthesia in 32 cases of tabes as regards the root areas. The regions least affected are seen to be the upper cervical, lower dorsal and upper lumbar, while those most affected are the fourth and fifth dorsal and fifth lumbar and first sacral. It is thought that probably the frequency of affection of these two regions is dependent upon a precarious blood supply. Microscopic examination of the spinal cord and roots readily explains the nature and distribution of the sensory disturbances : the area of the cord found to be most completely affected in tabes is the mid-dorsal and lumbo-sacral regions, while the region of the lower dorsal and upper lumbar roots is less affected.

*Sense of Position of Joints.*—Among 30 cases tested, this sense was found intact in 7—all early cases. In 21 cases, the toes were affected, in 11 cases the whole lower limb, in 9 cases the arm, especially the fingers and wrist. Mott's conclusion is that loss of this sense is a very important factor in the production of inco-ordination of movement and that it is associated usually, but not necessarily, with disturbance of cutaneous sensibility. He believes that this sense is due rather to alteration in tension of structures about joints than to alteration of contact of surfaces : thus, arthropathies may occur without loss of this sense and without ataxy, but he has not met with a case in which this sense was lost without ataxy, although he has seen several cases of ataxy without loss of this sense.

*Visceral Disturbances.*—Bladder troubles were stated to be present in about one-half of the cases of tabes, in 60 per cent. of the taboparalytic cases. Gastric crises, not infrequently the earliest symptom, were present in 21 out of 60 cases of tabes (36 per cent.), in 8 per cent. of taboparalytics. In all cases they were associated with the mid-thoracic anæsthesia, although by no means every case of anæsthesia in this region was accompanied by gastric crises. The relative frequency and early appearance of gastric crises points to the same tendency to degeneration of the afferent visceral nerves or their intraspinal terminations as is seen in the intraspinal terminations of the posterior roots supplying the skin of the mid-thoracic region. Rectal crises were found as an early symptom in 8 per cent. of cases ; laryngeal crises in 2 cases of tabes, 1 case of taboparalysis.

*Deep Reflexes.*—Absence of knee-jerk occurred in less than 70

per cent. of cases of tabes, when first seen. Among the asylum cases, the knee-jerks were found absent on both sides in 77 per cent., on one side in 16 per cent. The triceps-jerk was usually found absent when the knee-jerk was absent, but it undoubtedly disappeared later than the knee-jerk.

*Tonus.*—Diminished tonus was found in all cases of tabes except 8, all of which were in the preataxic stage. There was a distinct relationship between the degree of hypotonus and the ataxy. In asylum cases, the hypotonus was not so marked: with the onset of mental symptoms, the hypotonus as well as the ataxy diminished or even, in the first stage of the disease, largely disappeared.

*Romberg's Symptom*, so constant and prominent in tabes, was very marked in only 15 per cent. of asylum cases, moderately marked in 26 per cent., slight in 7 per cent.

*Diseases of the Bones and Joints.*—Ten per cent. of his cases of tabes showed arthropathy, more often women than men. Cases are cited to show that there is no doubt that hard occupations involving much use of joints predispose to this condition. The pathological changes found in the joints in 2 cases are described. It is still a moot point whether disease of the bones and joints is dependent upon a definite lesion of the nervous system: the fact that it does not occur in peripheral neuritis is against the view that it is due to disease of the peripheral nerves. In one case of experimental ligature of the posterior roots of the cauda equina, Mott found osteoporotic changes in the leg bones which he thinks may have been due to the ligature causing vasodilator excitation, with dilatation of the vessels of the bones, and consequent absorption and diminution of the mineral matter.

*Cerebral Symptoms.*—In taboparalysis, the onset of cerebral symptoms may be sudden and indicated by various kinds of seizures.

*Slight Congestive Attacks*, with temporary disturbance of consciousness, are often disregarded for long. *Epileptiform seizures* may be the first evidence of cerebral affection and may precede or succeed spinal symptoms. Of 60 cases of taboparalysis, 64 per cent. had such attacks. A condition of status epilepticus may occur with hyperpyrexia and death—often, however, the cause of the seizures and of death in such severe cases is an associated acute disease, such as pneumonia or dysentery. Mott thinks that the fits often indicate an increased irritability preceding decay or even death of the psychomotor neurons of the cerebral cortex. *Apoplectiform seizures*, of various degrees of severity, may precede by months or years all other symptoms. Transitory defects of speech are particularly suggestive of either general paralysis or syphilitic endarteritis.

*Mental Symptoms* occurred in about 10 per cent. of cases seen outside of asylums. The *prodromal* mental changes (changes of character and of temper, loss of self-reliance and of power of concentration, etc.) may precede or be associated with symptoms of cord affection; even at this stage, the brain would show definite microscopic changes. "*Acute mania*," of which four types are considered, is the most common mental symptom which brings a tabetic or taboparalytic into asylums. *Hallucinations* are of great importance. Many of these are rather of the nature of illusions, being excited by definite peripheral irritation; thus the various pains and paræsthesiæ, cutaneous and visceral, may be insanely interpreted and give rise to the delusions of persecution associated with electricity in the body, twisting of the bowels, poisoning of food, etc. Visual hallucinations occurred in a number of cases with optic atrophy, even where it had led to complete blindness, probably due to a morbid functional or organic disease of the ideation centre of vision, presumably in the angular gyri, which was excited by the irritation of its associated diseased perceptive centre or the peripheral structures (retinæ and optic nerves) connected therewith. Like auditory hallucinations, with which they were frequently combined, visual hallucinations were generally associated with, and tended to engender delusions of persecution. *Delusions* of persecution (electricity, poisoning, etc.) occurred in 28 per cent. of sixty asylum cases: in three-fourths of such cases, there were auditory or visual hallucinations or both; all, with one exception, had a hereditary history of insanity. These delusions occurred more in tabetic patients with associated insanity than in taboparalytics—in the latter, grandiose delusions supervened so frequently. The delusions of persecution, with hallucination of sight and hearing, have an organic basis in the taboparalytic and generally persist throughout the illness; the grandiose delusions seemed less persistent, were much less frequently associated with hallucinations of sight and hearing, and a hereditary history of insanity was comparatively less frequent. *Dementia* exists in all cases of taboparalysis, as in general paralysis; but, in Mott's experience, death takes place before there is advanced dementia more frequently in cases of taboparalysis than in ordinary cases of general paralysis, no doubt because a man who has already suffered from degenerative changes in the cord is in a lowered state of vitality. The dementia, generally speaking, is proportional to the extent of the brain atrophy (provided that the acuteness of the process has not been so great as to leave no time for absorption of the products of decay); and it is a fact that, in cases with *tabes dorsalis*, one finds as a rule less brain wasting than in an ordinary case of general paralysis.

*Notes of 74 cases* are given in great detail, with detailed patho-

logical results in many cases and numerous photographs and diagrams illustrating pathological changes, sensory disturbances, etc. These cases are divided into the following groups: (1) cases of tabes with some unusual clinical phenomena or adapted to explain some essential feature of the disease, 23 cases; (2) cases of tabes with insanity, 7 cases; (3) cases of taboparalysis, 21 cases; (4) cases of taboparalysis with marked speech affection, 6 cases; (5) optic atrophy and taboparalysis, 6 cases; (6) conjugal tabes and paralysis, 6 cases; (7) ataxy with non-progressive (? alcoholic) dementia, 2 cases; (8) interesting hospital cases, 3 cases.

*Morbid Anatomy and Pathology.*—The spinal cord lesions of tabes and taboparalysis as regards the affections of the posterior roots and posterior columns are, as a rule, identical. The *posterior roots* showed atrophy or disappearance of fibres, but none of the signs of Wallerian degeneration. Any changes found in the *spinal ganglia* were insignificant as compared with the atrophy of the posterior roots, and Mott is not satisfied that they were sufficient to account for the disease (Marie's theory). The same is true of the changes found in *peripheral nerves*, chiefly in advanced cases: this coincides with the obvious root-distribution of sensory disturbances observed *intra vitam*. In all the twenty-eight cases examined, the three sets of exogenous fibres of the *posterior columns*—proceeding respectively to the root zone of Charcot, the cells of Clarke's column and to form Goll's column—were affected, although in varying relative degree according to the relative amount of degeneration of particular roots. The ataxy, Mott thinks, has a decided relationship to the atrophy of the plexus around the cells of Clarke's column; he has seen no case in which this was pronounced without marked ataxy. *Lissauer's tracts* were in a few cases not affected at all, and in many cases but very slightly, when there was marked affection of the root-zone of Charcot. In 9 cases, with very marked ataxia, the descending *endogenous* system of fibres of the posterior columns was markedly affected in 6 cases and very markedly in 3; the ascending endogenous system was never completely destroyed throughout the cord. There was a decided relationship between the degree of affection of the endogenous systems of the posterior columns and the degree of ataxy—only in advanced bedridden cases was marked atrophy of these systems found, and there was none in the pre-ataxic stage. In advanced cases of tabes the *cells of Clarke's columns* may be degenerated, with consequent atrophy and sclerosis of the ventral and dorsal cerebellar tracts. By far the most frequent and important associated degeneration and subsequent sclerosis was found in the *pyramidal tracts* (in 20 out of 28 cases). The changes here are of two kinds—(1) well-marked acute degenerative changes, on one or both sides, which can be traced from

the cerebral cortex down to the pyramidal tracts, due to acute destructive changes in the cortex and associated clinically with epileptiform seizures; (2) primary degeneration, with sclerosis, which can be traced only up as high as the mid-dorsal region; it involves only the crossed pyramidal tract or tracts, and is occasioned by a slow progressive atrophy of the cortical psychomotor neurons with the longest axons—the fibres most affected being those most remote from their seat of nutrition, i.e. the leg fibres; those cases are characterised principally by a slow progressive dementia, without epileptiform seizures. In advanced cases of tabes, amyotrophy from affection of the *anterior horn-cells* may occur, especially in the small muscles of the hands and feet, probably due to total destruction of the sensory neurons which are in relationship with them; in such cases there was always a marked atrophy of the endogenous, as well as the exogenous fibres of the posterior columns. In 3 cases out of 28 there was *heterotopia spinalis*—a condition never seen by Mott in other diseases. In one of these cases there was active proliferation of the epithelium lining the dilated central canal and of the surrounding glia. The morbid changes in the *brain* in taboparalysis are essentially the same, but not so advanced as in ordinary paralysis. The brain-wasting was seldom great, and was most marked in the fronto-central regions. Thickening of the pia arachnoid and dilatation of the lateral ventricles were in proportion to the atrophy. The ependyma of the lateral ventricles was usually granular, but not in early cases. Granulation of the floor of the fourth ventricle was, however, always present, even in the earliest cases. This marked tendency to ependymal change, not found in other brain diseases, is regarded as a sign of irritation from the cerebrospinal fluid and as additional proof—like the filling up of the central canal of the cord—of biochemical toxic irritation. Changes in the *nerve-cells* are of two kinds—(1) a chronic atrophic process, beginning in the superficial layers of the cortex, very slow and insidious, frequently in isolated foci, without any marked inflammatory reaction; (2) an acute process, occurring in paroxysms and associated with the formation of an acute irritant toxin having a local action and causing, partly by its own chemical properties and partly by the inflammatory reaction of the vessels, an acute coagulation necrosis of the cells, usually, but not necessarily, accompanied by an overgrowth of glia cells and vascular changes. Atrophy of the tangential and supra-radial *nerve-fibres* was found in every case, mainly in the fronto-central regions and sometimes limited to the pre-frontal region. Frequently this atrophy was purely local, corresponding to local atrophy of cells. In advanced cases the inter-radial and radial fibres were atrophied. *Glia proliferation* is not described in detail. “The most constant and striking change which can be

observed in progressive paralysis is afforded by the appearance of *vessels*," viz., a proliferation of cells on the vessel walls and in the perivascular lymphatic spaces. A description of these cells, the plasma cells of Marscholko, is given. Mott is now of opinion that they are derived from lymphocytes, and not from endothelial cells, although the latter do undergo active proliferation. He thinks that these plasma cells are almost pathognomonic of general paralysis; they were present in all cases of general paralysis and taboparalysis examined, and not in any other condition, except in one case of Congo sickness and one case of multiple syphilitic gummata. They were found only in the brain, and more especially in those regions which generally show wasting in general paralysis. Their existence in abundance is clearly associated with the amount and intensity of the acute neuronc irritation and destruction, and they are indicative of an acute irritative process.

With regard to the pathology of taboparalysis in general, Mott's conclusion is that "the clinical symptoms associated with the anatomical findings undoubtedly show an intense irritant morbid process, followed by acute neuronc destruction." The irritant is, presumably, of a biochemical nature and produced paroxysmally by a *localised conspiracy of factors*; the presence of the plasma cells and the proliferation of the glia, lymphocytes and endothelium are the signs of the local vital reactions of the tissues to this poison. "The paroxysmal character of seizures, followed by apparent betterment, and the microscopical evidence of an intense irritative and destructive process more obvious than in almost any other brain disease, point to the *formation in the blood of a toxin which fixes on to certain portions of the central nervous system*, which either stress, heredity or anatomical conditions place in a lowered state of resistance." The morphological conditions of the arterial and venous circulation in the fronto-central regions, favouring anæmia and venous congestion, render these regions specially liable to suffer. On the analogy of diphtheria and tetanus, one is warranted in supposing that the syphilitic virus may convey several poisons, one of which may be latent, and—operating only under certain abnormal metabolic conditions of the neurons (*e.g.* due to stress, heredity, etc.)—may produce these late manifestations, tabes and general paralysis.

The paper concludes with notes on sensory disturbances of the skin, deep structures and viscera, and also on the subject of co-ordination and inco-ordination. The rôle played in co-ordination by the spinal, cerebellar and mesencephalic, and cerebral neurons is shortly discussed. In both tabes and general paralysis there is a failure in co-ordination, but the cause is different; in the former the coarse subcortical reflex and semi-automatic adjustments are at fault; while in the latter, the fine cortical adjustment fails.

The essential cause of tabes is undoubtedly the degeneration of the posterior roots, but marked ataxia does not occur without some degeneration of the endogenous systems. Mott's conclusion is that the afferent fibres to the cerebellum by way of Clarke's column plays a specially important part in co-ordination, and that the impressions coming from the deep structures are of more importance in co-ordination than those from the superficial structures. In man, co-ordination is primarily but not entirely cerebral. Assuming with von Monakow that there is a special efferent tract from the cortex, which regulates reflex tonus and adjusts the reciprocal innervation of functionally correlated groups of antagonist muscles (Sherrington), we can understand that, as this reflex tonus is progressively abolished in tabes by the destruction of the posterior roots, there is a disturbance in the balance of this reciprocal cortical innervation of the spinal neurons, and the resulting inco-ordination is partly due to uncontrolled over-action of one system of cortical psychomotor neurons. Ataxy is due not only to loss of function from neuronie destruction, but to physiological over-action of structures, which normally act in opposition to those which are destroyed. In this fact may be found an explanation of the diminution or disappearance of ataxy in some cases of taboparalysis, when signs of cortical degeneration appear—the cortical disease withdrawing the possibility of over-action. So the effect of cortical disease in withdrawing the inhibitory influence which the cerebral cortex normally exercises on reflex tonus, may explain the reappearance of the knee-jerk in certain cases.

A. W. MACKINTOSH.

**ON THE SYPHILITIC NATURE OF TABES.** *Bull. Méd.*, May 9, (229) 1903, p. 441.

At a meeting of the Société Française de Dermatologie et de Syphiligraphie, held on April 20th, 1903, a discussion took place on this subject.

M. Audry held that the relationship of tabes and syphilis was now admitted by all. The only question was whether tabes was a syphilitic or a parasyphilitic affection. Of the arguments in favour of the latter view, he thought that that of the rarity of the occurrence of true syphilitic symptoms in tabes was most important. Under mercury, he had seen absolute arrest of the progress of the disease, and improvement in certain of the symptoms, viz., in the gait, in vision, and in bladder troubles. No effect, however, was ever produced upon the reflexes. Benefit following mercurial treatment did not necessarily indicate syphilis, since lupus, epithelioma, etc., often improve under mercury. Of the various methods, inunction was the one he preferred.



M. Milian believed that specific treatment in tabes was absolutely useless. According to Nageotte, the early stage of tabes would show meningeal inflammation. Milian, however, had performed lumbar puncture before and after specific treatment, and had never observed any diminution of the leucocytosis.

Fournier pointed out that where nervous lesions occurred early in syphilis, ordinary specific manifestations were often present. Still, their coincidence with tabes was rare. The effect of specific treatment in tabes had lately been greatly exaggerated. He had never seen a single case of cure either in tabes or in general paralysis. Some symptoms had been benefited, but he had never observed any improvement in the lightning pains or in the reflexes, nor seen cure occur either in optic atrophy or in the less common tabetic exostosis.

A. A. SCOT SKIRVING.

**A CASE OF PROGRESSIVE MUSCULAR ATROPHY OCCURRING  
(230) IN A MAN WHO HAD HAD ACUTE POLIOMYELITIS  
NINETEEN YEARS PREVIOUSLY. CHARLES S. POTTS, *Univ.  
of Penna. Med. Bul.*, March 1903.**

THE patient, a man, aged 22, was a cigar-maker by occupation. When seven months old he had an illness attended with convulsions, which left him paralysed in the right leg, and partially so in the left leg and right arm. The left arm was unaffected and remained so till three years ago; but during these past three years it had been gradually getting weaker and he could no longer make use of it in his daily occupation.

On examining the left arm the atrophy was most apparent in the thenar and interossei muscles, but fibrillary tremors were well marked in most of the muscles of the arm. These fibrillary movements were also present in the muscles of the right arm. The right leg was typical of infantile paralysis. It was quite useless, and two inches shorter than the left. The movements in the left leg were fairly good, excepting dorsal flexion of the foot and toes.

The author gives in abstract 37 other cases of progressive muscular atrophy following infantile paralysis, but these are all the cases he can find after an extensive search through medical literature.

W. K. HUNTER.

**REPORT OF TWO CASES OF BULLET INJURIES TO THE LEFT  
(231) LATERAL HALF OF THE UPPER PORTION OF THE  
SPINAL CORD. J. T. ESKRIDGE and E. J. A. ROGERS,  
*Journ. Nerv. and Ment. Dis.*, March 1903, p. 129.**

IN this memoir the authors describe the signs present in two cases of spinal cord injury, both of which were operated upon; the first case died and the results of the autopsy are given.

The first patient was shot from in front through the neck, and at once fell down and could not rise. An examination nearly two days later showed that the left arm and left leg were completely paralysed, and the right limbs were weak. Tactile sensation was more or less blunted over the whole of the left arm and hand, but not elsewhere; whilst pain and temperature sensations were much diminished or absent over the right limbs and right side of the trunk, and over the left shoulder. On the other hand, joint sensation was perfect everywhere, and "posture sensation" was normal in the right limbs but lost in the left ones. The right pupil was twice the size of the left, the latter being apparently normal in size; there was slight ptosis of the left upper eyelid. The knee-jerk was normal in the right leg, but absent in the left.

Laminectomy was performed four days after the injury, and a spicule of bone which had been driven into the left lateral region of the cord at the fifth cervical level, was removed. The patient died a few days later, and it was then found that the cervical part of the spinal cord was inflamed and softened. The spicule of bone had not penetrated more than about half way from the surface to the central grey matter.

In the second case, a youth of 19 was shot from behind, and he at once fell down and was found to be paralysed. Examined five hours later, it was found that there was incomplete paralysis of the left arm and leg, the knee-jerk being brisk on the right side and increased on the left. There was loss of tactile sense over the left arm, and of pain and temperature senses down the right side. Muscular sense was absent in the left leg and hand. No ocular signs were present. Laminectomy was performed at once, and a bullet was found pressing upon the spinal cord in the lower cervical region, the theca and cord being intact. For a few days after the operation both knee-jerks were absent, and the left remained diminished for several weeks after the right had become normal. Complete recovery ensued in about six months.

In neither of the cases are the localising signs clearly given, and the anæsthesias charted, especially in Case 1, do not correspond to the segmental areas usually accepted. The chief value of the cases is in confirming the fact that in the cervical region a pressure from outside the theca can produce symptoms practically identical with those of a hemi-section of the cord, *i.e.* paralysis of the limbs of the same side, and of loss of pain and temperature senses of the opposite side. Owing to the inflamed state of the cervical cord of the first case it was impossible to identify the tracts that had been severed.

STANLEY BARNES.

**TRAUMATIC LESIONS OF THE SPINAL CORD WITHOUT  
(232) FRACTURE OF THE VERTEBRÆ.** WILLIAM G. SPILLER,  
*Univ. of Penna. Med. Bull.*, Feb. 1903.

IN this memoir the author gives a careful account of a patient who fell a distance of eight feet on to his face. He was unconscious for several hours, and two days later (when the mental condition was good) was found to have great general weakness of both arms, considerably impaired movement of the legs, and incontinence of urine and fæces; the knee-jerks were present but not exaggerated, and the plantar reflexes were uncertain. Sensation for touch was normal, but for pain and temperature was deficient below the neck. There was no sign of vertebral fracture. Seven days later, the left knee-jerk was absent and the right was feeble; both plantar reflexes were of the extensor type. Much recovery of power in the legs occurred and a little in the arms, but he died thirty-eight days after the accident.

At the necropsy there was no sign of fracture of the vertebræ nor of pressure on the cord, but in the fifth cervical segment there was found a transverse myelitis with minute hæmorrhages and Marchi degeneration scattered throughout the sections. Ascending and descending degenerations from this segment were well-marked, the crossed pyramidal tracts being severely degenerated. Slight cytological changes were found in the anterior horn cells of the lumbar enlargement.

The author discusses the fibre-paths for touch, pain and temperature sensations, and leans to the view that all sensory fibres may transmit tactile impressions. He also discusses the character of the lesion, especially as distinguished from hæmatomyelia, and further suggests that some cases of "traumatic hysteria" are in reality dependent upon an organic change in the brain and cord. He does not indicate any cause for the behaviour of the knee-jerks in this case.

STANLEY BARNES.

**A CASE OF INTRA-DURAL SPINAL CYST, WITH OPERATION  
(233) AND RECOVERY.** By W. G. SPILLER, J. H. MUSSER, and  
EDWARD MARTIN, *Univ. Penna. Med. Bull.*, March 1903.

IN this communication the authors describe the clinical condition of a woman aged twenty-six, who had suffered attacks of pain in the region of the left hip-joint for several years. This pain, at first slight and intermittent, gradually increased in constancy until she first came under observation in January 1902. It was then found that the pain was felt in the anterior part of the left thigh, over each sacro-sciatic foramen, and in the lumbar region on both sides of the spinal column. No other definite signs were then present, but when again seen in June 1902 there was

numbness of the left foot, and occasionally the left leg would give way in walking; slight disturbance of tactile sensation below the knee, absence of the left knee-jerk and ankle-jerk and a tendency to contracture in the left lower limb were noted, but there was no atrophy or paralysis. A few days later the flexors of the hip became weak, and both knee-jerks were lost. An operation was decided upon, and on June 30th Dr Martin removed the laminae of the first three lumbar vertebrae; the dura was incised and there presented a thin-walled cyst whose removal was followed by a gush of pent-up cerebro-spinal fluid. The patient rapidly recovered, and in March 1903 was practically well. The cyst was supposed to be non-parasitic.

The account of this case is followed by "A brief report of eleven cases of tumour of the spinal cord or spinal column," by Spiller. He concludes, "My experience makes me very careful in giving a favourable opinion regarding operation in cases of tumour of the spinal cord or vertebrae, and yet in view of the hopelessness of treatment without operation I believe that operation should be attempted in every case of spinal tumour which seems suitable."

STANLEY BARNES.

**A REPORT OF TWO CASES OF MULTIPLE SCLEROSIS WITH  
(234) NECROPSY. WILLIAM G. SPILLER, *Am. Journ. Med. Sc.*, Jan.  
1903.**

ACCORDING to Dr Spiller multiple sclerosis appears to be rather a rare disease in America, and only three previous cases with post-mortem results have been reported in that country. Of the two cases here described one was typical of the disease both clinically and pathologically, while the other was characterised clinically by the unusual occurrence of marked muscular atrophy and absent knee-jerks, and pathologically by the presence of changes in the cells of motor nuclei and of secondary degeneration in the crossed pyramidal tracts.

CASE I. Male, aged 25 years. Typhoid at 18. Excess in alcohol and tobacco, but no venereal disease. After great exposure to cold and damp in the winter of 1897-98 patient suddenly experienced stiffness in the lower extremities and pains in the thighs. His condition fluctuated with occasional loss of sphincteric control until November 1900, when he presented the following signs. Scanning articulation, nystagmus, optic atrophy, tremor of tongue, intention tremor in right arm, spastic and ataxic paraplegia with exaggerated deep reflexes, ankle clonus and extensor responses. The patient died in December 1901 with general emaciation and glycosuria. The microscopic examination of the brain and spinal cord revealed numerous areas of sclerosis, especially in the cord.

**CASE II.** In 1891 a woman 41 years of age, who denied venereal disease and alcoholic excess but admitted excesses in tobacco, after residence in a damp house complained of numbness in the legs and progressive loss of power in the lower limbs with staggering gait. She rapidly lost all power in her legs and all control over her sphincters, but her condition improved again and she was able to walk with difficulty until 1899. In 1901 her articulation was drawling, her lower limbs were completely paralysed, atrophic and spastic. Knee and Achilles jerks were absent; plantar responses were extensor in type. Forearms and hands atrophied and almost powerless. Almost complete external ophthalmoplegia and partial atrophic cupping of both optic discs, but normal pupillary reactions. Sensation normal. Incontinence of urine and faeces. Death in July 1901.

Microscopic examination revealed numerous sclerotic areas throughout the brain and spinal cord. One such area implicated the nuclei of the oculomotor, trochlear and abducent nerves as well as the posterior longitudinal bundles. A sclerotic area was found in the left optic nerve, but the right appeared to be normal. Secondary degeneration of moderate intensity was present in each crossed pyramidal tract below the mid-thoracic region, but was more marked on one side. Sclerotic areas involved the posterior root fibres after their entrance into the posterior columns in the mid-lumbar region. In the lower lumbar and sacral segments the disease affected the anterior horns and changes were evident in some of the ganglion cells. A branch of one of the plantar nerves was partially degenerated. In conclusion the author of the paper points out how the morbid anatomy of the case explained the unusual clinical phenomena and refers to other records of similar atypical cases of multiple sclerosis which are to be found in medical literature.

E. FARQUHAR BUZZARD.

**A CASE OF PARALYSIS OF THE VERTICAL MOVEMENTS  
(235) OF THE EYEBALLS, WITH CONSERVATION OF THE  
LATERAL MOVEMENTS.** ALEXANDER BRUCE, *Trans. Med.-  
Chir. Soc. Edin.*, vol. xix. p. 206.

**AUTHOR** describes a case of a single woman, aged 46, who, after severe exposure, had a rigor, and suffered from pain, deafness and tinnitus in the left ear, with weakness of sight and slight headache. These symptoms were followed by a peculiar forced movement, in which there was a constant tendency to fall backwards, or even to bear backwards against a supporting hand, from whatever position she was placed in, so that walking and sitting became impossible. Accompanying ocular symptoms were at first as follows: loss of the upward movement of the eyeballs and upper eyelids in at-

tempting to look upwards; conservation of the conjugate downward and lateral movements; slight diminution of convergence; no ptosis; no alteration of pupils; slight hyperæmia of fundus oculi. Exaggeration of knee-jerks, ankle clonus and patellar clonus, but no other motor or sensory anomaly. Then followed occasional vomiting, increasing hebetude, dilatation and immobility of pupils. On the day of death ptosis developed.

Autopsy revealed an angio-glioma affecting the grey matter round the aqueduct of Sylvius, from a point midway between the two corpora quadrigemina to the third ventricle, into which it projected (Figs. 1, 2, 3 and 4). Tumour had spared the fourth nucleus and lower part of the third nucleus. A second small tumour was found on the septum lucidum. Slight distension of both lateral ventricles with fluid.

Author refers to cases by Gowers (*Trans. Ophth. Soc.*, i. 81, p. 117), Wernicke (*Berlin klin. Wchnschr.*, 1878, p. 154), Lang and Fitzgerald (*Trans. Ophth. Soc.*, ii. p. 230), in which somewhat similar symptoms were produced by lesions in the inferior vermiciform process of the cerebellum, in the optic thalamus and corpus striatum.

AUTHOR'S ABSTRACT.

**BILATERAL GLIOMA OF THE CENTRUM OVALE.** E. LUGARO, (236) *Riv. di Patol. nerv. e ment.*, f. 2, 1903, p. 49.

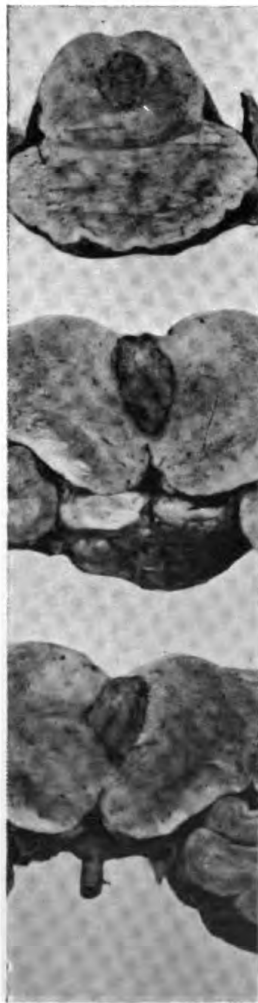
THE history was as follows: A woman, aged 38, of robust constitution and who had until four and a half months previously enjoyed good health, was admitted to the hospital in a state of profound mental stupor, interrupted by hallucinations. The pupils reacted slowly, her knee-jerks were exaggerated, there was spasmodic paresis of the lower limbs, incontinence of fæces and of urine. She rapidly became worse, collapsed and died. At the autopsy, on cutting into the brain on the left side a yellowish tumour mass was seen—about the size of a five lire piece, and without clear boundaries—lying in the centrum ovale. Above, it did not invade the grey matter, but below it spread into the internal capsule and basal ganglia. On the right side the white substance was of normal colour but of denser consistence. The corpus callosum was normal.

Pieces of tissue were taken from both affected areas and fixed in Form. ten per cent. Sections from the tumour were stained by toluidin blue, Apathy's hæmatin method and Weigert's method. The cells of the central convolutions were also examined.

Left side: In the tumour substance there were many giant cells of different size with many polymorphic nuclei. Scattered amidst these cells were found young actively proliferating neuroglia cells. It is from these cells that the giant cells are formed by direct division of the nucleus without division of the protoplasm.



FIG. 1.—To show tumour projecting into the third ventricle.



FIGS. 2, 3, 4. — Cross sections through upper pons and cerebral peduncles to show position of tumour.





The nuclei so formed may have polymorphic appendages, and when numerous are arranged round the periphery of the cell. Some of the largest cells showed vacuoles, and in some of the largest nuclei there were pale coloured drops.

The author is convinced of the neuroglial origin of the giant cells. By Weigert's method the neuroglia fibres showed little relation to the cells. Very few astrocytes were to be seen.

Around some capillaries, smallest arteries, and veins, was seen an enormous quantity of small neuroglia nuclei, some round and dark, others large and clear and undergoing direct division.

Right side: Here the giant cells were few in number, the predominant type being cells with a more or less large reticulated nucleus with nucleolus and at times surrounded by protoplasm. In some pieces of the tumour the nuclei were very dark and traversed by sinuous clear tracts containing very few nuclei which hardly stained with the hæmatin. These tracts were seen to be composed of capillaries surrounded by degenerated neuroglia, whose fibres could be seen to be continuous with the surrounding neuroglia but had lost the affinity for the stain and had become agglutinated into a homogeneous mass. In some parts of the tumour the fibres were finer and relatively scarcer. Occasionally true astrocytes with protoplasmic processes and differentiated fibres were seen.

In the cerebral cortex there was no increase of neuroglia but the giant nerve cells, here and there, showed the "reaction à distance." Other cells were pale and diffuse.

Lugaro thinks that the giant cells with one nucleus and abundant protoplasm represent degenerative forms of neuroglia and are purely pathological, probably being destined for destructive purposes. The production of abundant neuroglia fibres is to be ascribed to the work of the cells which retain the characters of the normal neuroglia cell. He excludes the possibility of the tumour being formed from aberrant epithelial cells, the cells rich in protoplasm being simply transformed neuroglia cells. The spasms observed in the patient were due to lesion of part of the pyramidal fibres, as shown by the reaction of some of the giant nerve cells. The abnormal excretions from the pathological neuroglia exercised a toxic influence on the nervous system as shown by the diffuse chromatolysis in some of the cells above noted.

DAVID ORR.

**SPECIFIC AUTOCYTOTOXIN AND ANTIAUTOCYTOTOXIN IN  
(237) THE BLOOD OF EPILEPTICS.** CARLO CENI, *Neurol. Centralbl.*, April 16, 1903, p. 338.

THE author records that he had observed that the repeated injection into epileptics of blood serum drawn from other epileptics

had in some instances an unfavourable and in others a beneficial effect on the condition of the patient.

The injection into epileptics of 1-3 c.cm. of the serum of rabbits immunised against the serum of epileptics was constantly followed by a local and a general reaction, the latter being of a specific character, and consisting in an elevation of the temperature not exceeding 39° C. and frequently in a condition of status epilepticus. These phenomena were not induced by injection of this serum into non-epileptics, by injection, whether into epileptics or non-epileptics of serum drawn from rabbits inoculated with normal human serum, nor by injection of normal rabbit's serum into epileptics.

The author consequently assumes that the blood of epileptics contains specific toxic substances—autocytotoxins—the product of abnormal metabolism. The autocytotoxin of the epileptic consists of two elements: (a) cytase, alexin, sensitive substance, or complement; and (b) antibody, intermediate body, or sensibilising (thermostable) substance. The serum of an epileptic, when injected into rabbits, is said to lead to the production in them of sensibilising substance. This statement of the author's must be accepted with some reservation, for one would expect that the injection into animals of serum containing a sensibilising substance would lead to the appearance of an anti-sensibilising substance in the serum of the treated animal. The cytotoxic serum drawn from the immunised rabbits was still active after it had been heated to 56° C.: the sensibilising substance is thermostable. Other rabbits were inoculated with epileptic serum which had been heated to 56° C., and the serum of these rabbits was found to be but feebly toxic for epileptics.

The specific toxic phenomena observed in epileptics after injection of serum derived either from other epileptics, or from animals inoculated with epileptic serum, are due, not to the further introduction of cytotoxin, but to the sensibilising substance of the cytotoxin which renders the epileptogenic elements more susceptible to the alexin already circulating in the epileptic organism. The serum of epileptics may be assumed to contain a relatively small amount of sensibilising substance, and thus toxic symptoms are not always produced in epileptics after the use of this serum. The serum of prepared rabbits, however, contains the sensibilising substance in much greater amount, and can therefore induce marked toxic effects in every epileptic.

The toxicity of the cytotoxic serum derived from the prepared rabbit could be in great part, or entirely, neutralised by the addition of ordinary epileptic serum, which consequently contains a specific antiautocytotoxin—probably an antialexin. The reinjection into epileptics of their own serum was in some instances followed by considerable improvement.

W. T. RITCHIE.

**THE PROGNOSIS AND CURABILITY OF EPILEPSY.** W. ALDREN  
(238) TURNER, *Lancet*, vol. i., 1903.

1. A total of 366 cases, chiefly derived from the Out-patient records of the National Hospital for the Paralysed and Epileptic, have been used for the investigation.

2. Only cases of genuine idiopathic epilepsy, which had been under constant observation and treatment for a period of at least two years, have been taken. All cases of "symptomatic" epilepsy, or cases otherwise complicated, were as far as possible eliminated.

3. The cases have been divided into three series, according as they have responded, successfully or otherwise, to treatment—arrested, improved, and confirmed cases. The influence of the various conditions modifying prognosis has been mentioned in detail, the results of the observations being stated in percentages.

4. A family history of epilepsy will be found more frequently amongst those who have become confirmed epileptics, but an hereditary history of epilepsy does not necessarily militate against the prospects of arrest or improvement of the disease in any given case.

5. The age at the onset of the disease has an especial bearing upon the prognosis. The most unsatisfactory cases are those in which the disease commences under ten years of age; they show the smallest percentage of recoveries and the largest of confirmed cases. If the disease arises between 15 and 20 years of age, an almost equal percentage of arrested and confirmed cases may be expected. The greatest percentage of confirmed cases is found amongst those in whom the disease begins between 25 and 35 years of age, from which time onwards there is a steady increase in the expectations of arrest and diminution in the number which become confirmed.

6. The duration of the malady influences the prognosis to the extent that arrest, or improvement, are much more likely during the first five than during the second five years. Cases may, however, be arrested even after a duration of from twenty to thirty years.

7. The greatest percentage of confirmed and the lowest percentage of arrested cases occur in those epileptics who are subject to daily or weekly attacks, while conversely the smallest percentage of confirmed and the highest of arrested cases occur in those whose fits are as infrequent as once or twice a year.

8. The character of the seizures influences the prognosis to the extent that the major attacks are the most tractable; then follow combined major and minor seizures; and lastly, the minor attacks occurring alone.

9. Marriage exerts little, if any, influence upon epileptic fits.

Some patients are relieved; others are made worse. In the majority of cases the disease remains unaffected.

10. Pregnancy has little influence upon the seizures; at the best there may be only a temporary respite. On the other hand, the puerperium would seem to be especially favourable for the recurrence of fits; while lactation also is not without an exciting influence in their production.

11. The common incidence of epileptic fits is an irregular periodicity. There are types, however, which have been described as "increasing" or "decreasing," according as the fits increase or decrease in number in a definite period of time, or in which there is a shortening or lengthening of the intervals between the fits. A case of increasing type may by treatment be converted into one of the decreasing variety.

12. Long remissions, induced either by successful treatment or from spontaneous cessation of the fits, sometimes lasting for several years, are not unusual in epilepsy; they are of favourable prognostic value, but are not synonymous with a cure of the disease.

13. From the collected statistics a period of arrest for at least nine years has been fixed as the basis upon which a cure of epilepsy may be established. With this definition of a cure the writer regards 10.2 per cent. of epileptics as curable.

14. There are some cases of epilepsy which may be regarded as belonging to a curable type of the disease. These present little or no mental impairment, notwithstanding that fits may have existed for a long period. In the cases in which arrest took place, cessation of the fits occurred within the first year of continuous treatment in over 50 per cent.

AUTHOR'S ABSTRACT.

**ON RABIES.** G. ANGIOLELLA, *Il Manicomio*, 1902, f. 3. (239)

THE author relates the case of a boy of 3½ years who died about two months after having been bitten on the face by a rabid dog. Treatment by Pasteur's method was employed three weeks after infection, but was presumably undertaken too late to do more than modify the symptoms of the disease, which began to manifest themselves about a month later.

The brain, on microscopic examination, showed congestion, acute periarteritis and disintegration of nerve cells. In the spinal cord there was a more marked periarteritis, whilst the nerve cell lesions were less advanced. Changes of an essentially similar character were present in the spinal and sympathetic ganglia. Four rabbits, inoculated with the cerebro-spinal fluid and an emulsion of the spinal cord and medulla, died in from two to three days with typical symptoms of rabies. They presented nerve cell and vascular lesions similar to those found in the boy.

Remarking upon the case, Angiolella specially notes the fact that the nerve cell lesions were most advanced in the frontal lobes. This he explains by reference to experimental observations, which show that the virus of rabies is propagated along the nerve trunks, and that there is special involvement of those portions of the central nervous system that are anatomically connected with the nerves of the part primarily infected. In this case the Schneiderian membrane was lacerated, and he supposes that the virus passed directly to the frontal lobes. He discusses the significance of the histological lesions, and, in opposition to some other observers, takes the view that neither the nodular cell-infiltration in the brain and cord, nor the proliferative changes in the cell capsules of the sensory ganglia, have any specific character. The lesions differ from those of other acute infections only in degree, and not in nature. He considers that the morbid changes are diffused throughout the nervous system and not specially localised in the bulb or in the cranio-spinal ganglia, as some maintain.

W. FORD ROBERTSON.

**THE ETIOLOGY OF SLEEPING SICKNESS.** Preliminary note.  
(240) ALDO CASTELLANI, *Lancet*, March 14, 1903, p. 723.

**ON THE ETIOLOGY OF SLEEPING SICKNESS.** A. BETTENCOURT,  
(241) A. KOPKE, G. DE RESENDE, and C. MENDES, *Brit. Med. Journ.*,  
April 18, 1903, p. 908.

CASTELLANI claims to have found the streptococcus of sleeping sickness, classing it between *s. pyogenes* and *s. lanceolatus* (Fränkel's diplococcus). He could not find any germs mentioned by other workers. This streptococcus was obtained from the blood and cerebro-spinal fluid in 9 out of 11 autopsies; *in vita*, once from the blood and in two cases out of three from the cerebro-spinal fluid. Its appearance varies, depending on the media on which the germ has been cultivated. All transitions from long chains to typical diplococci may be seen. In old cultures the organism shows involution forms, being sometimes club-shaped. It stains easily with the ordinary solutions of the aniline dyes. It grows well on gelatine, thus differing from *s. lanceolatus*, and more vigorously on agar than *s. pyogenes* and *s. lanceolatus*. Unlike them it does not coagulate milk. It is a facultative anaerobe. In his few experiments he found that agglutination was caused by the blood of patients with sleeping sickness, in contrast to Broden's bacillus. The Portuguese state that their diplococcus fails to grow on the usual media and they make no mention of agglutination. The author admits that his experiments are as yet neither complete

nor sufficiently extensive, but he considers they point to this streptococcus being the cause of sleeping sickness.

The Portuguese Commission compare their researches with those of Castellani, who evidently had not seen their reports of September 1901 and August 1902, adding to and correcting their previous announcement. They deny any difference between their "hypnococcus" and Castellani's streptococcus, and the descriptions of each coincide in their main features. The Portuguese have evident priority, but the importance of discoveries so agreeing, made independently in distant and different regions of Africa, cannot be minimised, as they undoubtedly clear up the question of the cause of sleeping sickness.

C. H. G. GOSTWICK.

**ON THE DIAGNOSTIC VALUE OF THE POSITION OF THE  
(242) HEAD IN CASES OF CEREBELLAR DISEASE. FREDERICK  
E. BATTEN, *Brain*, Spring 1903, p. 71.**

THE author refers to Risien Russell's experiments upon the effects of ablation of one lobe of the cerebellum, and points out that in consequence of this lesion the trunk is curved with the concavity to the side of the cerebellar lesion, the side of the face is approximated to the shoulder on the same side, and there is a rotation of the neck so that the side of the face corresponding to the cerebellar lesion is turned upwards, and the chin is directed to the unaffected side.

The following questions are then raised :—

(1) *Is a definite attitude of the head assumed in cases of cerebellar disease?*

Of six cases of cerebellar disease examined pathologically by the author, a characteristic position of the head was present in three.

(2) *Does the position correspond to that produced by experimental lesions, and, if so, can the sign be used as a symptom of diagnostic value?*

The author admits that it is difficult to say in many cases of cerebellar tumour whether the position assumed has been due to the lesion itself or to pressure on structures in the immediate neighbourhood. He describes a case, however, which appears to be free from this latter objection.

The patient, a child aged 5, "when standing or sitting held her head to one side, so that her left ear was approximated to her left shoulder; her face was turned to the right, and the chin was slightly elevated." The position of the head is shown in a photograph. The child died in August 1902, four years after the first cerebral symptoms had appeared. For many months before her

death the intracranial lesion had apparently been quiescent, and had given rise to no symptoms.

At the post-mortem a very recent tuberculous meningitis was found, and a hard calcareous tumour, about the size of a hazel-nut, in the substance of the right lobe of the cerebellum. The tumour was adherent to the petrous temporal bone. There was no ventricular distension. The position of the tumour is shown in a photograph.

In three additional cases of cerebellar tumour in which there was an abnormal position of the head, in all the right ear was approximated to the right shoulder, and in all a lesion of the left lateral lobe was found at the post-mortem.

(3) *Is this position assumed in cases of intracranial tumour in which no gross lesion of the cerebellum can be found?*

The author, in answer to this question, describes a case of ventricular distension due to blocking of the foramen in the region of the fourth ventricle, in which, despite the fact that no tumour of the cerebellum was present, the head assumed the position above described.

The author summarises his conclusions as follows:—

*“Firstly.* A definite attitude of the head is not infrequently seen in cases of cerebellar disease in man, that position being with the ear approximated to the shoulder on the side opposite to the lesion, and with the face turned up to the side of the lesion.”

*“Secondly.* This position of the head, so far as the approximation of the ear to the shoulder is concerned, is the reverse, while the position of the face is the same as that seen after experimental ablation of one lobe of the cerebellum.”

“To answer the second portion of the question, viz., Can the sign be used as a symptom of diagnostic value? in the bare affirmative might lead to error, for the relative value of this symptom, in comparison with the other symptoms of a cerebellar lesion, is a question which needs most careful consideration in each individual case. It is probably a symptom of less importance than inco-ordination or weakness.”

*“Thirdly.* The fact that the position is sometimes present in cases in which there is no gross lesion of the cerebellum is a further reason for not attaching too great importance to the position assumed by the head.”

*“In conclusion.* It may be said that as an additional confirmatory sign of cerebellar tumour the position assumed by the head is of value, but too much importance should not be attached to its presence alone, or when opposed to symptoms which have been shown to possess greater diagnostic value.”

EDWIN BRAMWELL.

**RETINAL HÆMORRHAGES AS A DIAGNOSTIC FEATURE IN  
(243) FRACTURE OF THE BASE OF THE SKULL AND IN  
SUBARACHNOID HÆMORRHAGE. ROBERT A. FLEMING,  
*Edin. Med. Journ.*, April 1903, p. 297.**

THE material for this paper was derived from the post-mortem room, and was partly gathered during my term of office as one of the pathologists to the Royal Infirmary, Edinburgh.

I noted that in cases of subarachnoid hæmorrhage of fairly rapid development retinal hæmorrhages were found, and that if unilateral the eye in which they occurred was that of the side of the subarachnoid effusion. It has long been recognised that a subarachnoid, and even a sub-dural hæmorrhage may pass into the intersheath space of the optic nerve, and that in some cases optic neuritis and blindness have resulted; but so far as I can discover the incidence of retinal hæmorrhages and the particular eye affected have never been made use of in the diagnosis of subarachnoid hæmorrhage.

The following facts are important: The arachnoid divides the intersheath space surrounding the optic nerve into two parts, but it is not a complete membrane. The retinal vessels are a closed system, although there is free anastomosis at the papilla. The papilla is markedly constricted by the sclerotic ring. There is a very extensive perivascular system of lymphatics in connection with practically all the vessels of the optic nerve, and these lymphatics freely communicate with the intersheath space. Merely a gradual increase of pressure in these lymphatics is not sufficient to produce retinal hæmorrhages, but where a fairly sudden increase occurs, as in the majority of cases of subarachnoid hæmorrhage, retinal hæmorrhages result.

In some of my specimens the hæmorrhages may be seen not merely distending the pial sheath space, but actually entering the lymphatic channels at the apex of the space and passing between the fibres of the sclerotic coat—in one case nearly penetrating to the choroid. This explains the great increase of pressure at the papilla, and is the probable cause of the retinal hæmorrhages in these cases.

It is more difficult to account for unilateral retinal hæmorrhages in cases of subarachnoid hæmorrhage confined to the corresponding side of the brain. The subarachnoid space possesses several large cisterns, with one of which this paper has to do. The sinus basalis, situated just behind the optic chiasma and bounded posteriorly by the pons and on either side by the crura cerebri, has no median raphé excepting the stalk of the pituitary body which certainly divides the space anteriorly; posteriorly there is no division at all. How then does the blood



which often fills the whole of the sinus tend to pass down the corresponding intersheath space in cases in which there is a unilateral subarachnoid hæmorrhage? An analysis of the series of cases briefly referred to below proves that it generally does follow this course, and the explanation must be founded on hydrostatic principles. There was invariably found some flattening of the cerebral convolutions on the opposite side to the subarachnoid hæmorrhage. It may therefore readily be supposed that the sudden increase of pressure in the subarachnoid space must, if unilateral, tend to pass up the intersheath space of the same side while the opposite intersheath space may be occluded by the distorting force which must be associated with the production of flattening.

I have divided these cases into four groups.

GROUP I. contains five cases of fracture of the skull, in which the subarachnoid hæmorrhage was mostly unilateral, and retinal hæmorrhages were present but confined to the eye of the same side. All of these were cases of fracture of the base, excepting one, which was a fracture of the squamous portion of one temporal bone. In this particular case the fracture was on the left side, and the subarachnoid effusion on the right.

GROUP II. contains two cases of fracture of the base, in which the hæmorrhage was almost equally marked on both sides, and in which there were retinal hæmorrhages in both eyes.

GROUP III. contains five cases of fracture of the base, in which there were no retinal hæmorrhages visible to the eye after death. In three of these cases there was little subarachnoid effusion, or an effusion of apparently slow onset, and in one of the other two pressure seems to have interfered with the passage of blood into the intersheath spaces of the optic nerves.

GROUP IV. Naturally anxious to exclude any fallacy as to the retinal hæmorrhages being the result of the concussion, or the asphyxia, or other abnormal condition which might be associated with fracture of the skull, I determined to examine a number of cases of subarachnoid hæmorrhage which were not dependent on a preceding fracture, and selected a little group of four cases, in all of which subarachnoid hæmorrhage was present, and I found in three out of the four that there were retinal hæmorrhages; in one they were present in both eyes, in the other two cases in one eye only, and confined to the side of the chief subarachnoid effusion alone. The first case was one of pontine hæmorrhage, in which the blood had burst externally, filling the sinus basalis and both intersheath spaces with blood, and causing numerous retinal hæmorrhages. The other two cases were originally ordinary cases of cerebral hæmorrhage, which had, however, burst externally, and so had given rise to subarachnoid effusion more markedly on one side, and the retinal hæmorrhages present were confined to the eye of the

affected side alone. The fourth case of this group was also an ordinary case of cerebral hæmorrhage, which had almost ruptured into the ventricle, but only a very small amount of blood was found in the subarachnoid space, and it was in reality mostly blood-stained lymph. In this case there were no retinal hæmorrhages.

It is often no easy matter to diagnose a basal fracture, and a sudden subarachnoid hæmorrhage may occur as in the last group of cases apart from fracture. Retinal hæmorrhages are of diagnostic value in such cases, although they may not be of service in many instances from the point of view of operative interference.

AUTHOR'S ABSTRACT.

**PRODROMAL INSANE LAUGHTER.** CH. FÉRÉ, *Rev. Neurol.*, April (244) 15, 1903, p. 353.

**IMMODERATE** or insane laughter shows its relationship to madness not only in the intensity of its manifestation, its exceptional duration and its tendency to reproduce itself indefinitely, but above all in the unreasonableness of its causes.

It is the convulsive, boisterous, and prolonged explosion of some emotion which has no apparent cause, is often ill-timed and even altogether opposed to circumstances. The author proceeds to describe the phenomena which accompany its appearance, noting particularly the increased activity of certain glands—the lachrymal and salivary. No situations are able to arrest it; once it has begun it must go through to the bitter end. It is a sort of epilepsy which inevitably runs its course and completes the cycle of its convulsive manifestations. Excessive tension in general is a predisposing cause. When little children are ready to cry, some unseen circumstance may suffice at times to turn their tears into laughter, a fact which led Darwin to remark that these two channels may equally serve as outlets for an excess of nervous energy. The laughter seems related rather to excessive excitability than to excessive energy and often shows itself in conditions of fatigue.

All pathological conditions which accompany a widespread enfeeblement of the nervous functions and an increased excitability predispose to its occurrence. The author gives notes of two cases of chorea in which immoderate laughter was a premonitory symptom.

**CASE I.** The patient, a female, was brought at the age of 15 to the Bicêtre Hospital by her mother, who feared she was going mad. Following upon the death of her grandmother three weeks before, till when her behaviour had been normal, she had developed an insane laugh which for a week had appeared in the most

inappropriate circumstances, as in church or at the cemetery. She recognised that her joy was ill-timed, but explained it by motives of the most futile nature, *e.g.* that the cat was eating its tail or that a passer-by had his hat on squint. The fits of laughter often lasted 15 minutes or more. In a few days signs of chorea appeared, and in a short time the clinical picture was complete. Various hysterical complications arose at different times subsequently, and eight years later when the patient was pregnant the laughter again made its appearance at the 4th month, and was followed 12 days afterwards by choreic movements. The chorea lasted for 2½ months and disappeared in one night, apparently as the result of fright.

CASE II. Chorea preceded by insane laughter in a child after the injection of anti-diphtheritic serum.

The patient, a boy aged 12, born of healthy mother and alcoholic father, had been subject to migraine which began at 7 years and lasted till 11.

The serum was injected for the purpose of immunisation, the boy having been in contact with a case of diphtheria. There were no distinct physical reactions, but a striking moral change occurred. From being somewhat apathetic and unemotional, the boy appeared distracted and laughed interminably at the most insignificant things, and any attempt to show the absurdity of his behaviour only increased the laughter. This continued for 12 days, when choreic movements appeared in the face and tongue and soon spread to the limbs. The bursts of laughter diminished in number and intensity as the movements became more marked, but they did not altogether disappear and recurred from time to time for two months.

The author gives further notes of two cases in which insane laughter appeared as a premonitory symptom in organic cerebral disease.

In the first case, hemiplegia of the left and then of the right side was preceded by bursts of insane laughter which began to occur some months before the cerebral lesions were apparent.

The second case was that of a man aged 64, suffering from arterio-sclerosis, probably due to alcoholic excess, who, four months before a left-sided hemiplegia occurred, suffered from insane outbursts of laughter which were followed by an irresistible desire for sleep.

T. C. MACKENZIE.

### PSYCHIATRY.

**THE PHYSICAL SIGNS OF DEGENERACY.** VASCHIDE and VURPAS, (245) *Ann. di Neuro.*, Feb. 1, 1903.

THIS is the first of a series of papers which the authors intend to publish on the subject of degeneracy.

A definition of degeneracy is at present impossible, but included under this term are "those subjects who are born abnormal, different from others, remain so all their life, and die abnormal."

In this present article there is a comprehensive description of the physical lesions and malformations which are now recognised as stigmata of degeneration.

These lesions in the cases of the inferior degenerates (idiots, cretins, and imbeciles) attack both the somatic and mental development of the patients, and by some authorities the somatic disturbances are considered to be the determining cause of the mental deficiency. It is in these that we see the malformations and deficiencies of the nervous system and its enveloping structures which are so characteristic of the physical and moral type described by Morel, in fact it is only in these inferior degenerates that anything like a characteristic type can be seen.

As we rise in the scale of degeneracy we find that the somatic lesions become less prominent, until in the superior degenerate we notice chiefly the psychic disturbances, mental stigmata which bear to the mind the same relation that physical stigmata do to the body. But throughout the series, although the somatic stigmata may not be considered to be the cause of the mental deficiency, there is a close correlation between the morphological malformations, and especially those of the cranium and face, and the mental deficiency of the subject.

Besides these external somatic stigmata with which we are familiar, the authors assert that there are stigmata of degeneration in the internal viscera, which are quite as important, but which, up to the present time, have been generally neglected. These will be dealt with in a later paper.

With regard to the distinction between degenerative and purely pathological malformations, they assert that no line of demarcation can be drawn. They consider all these stigmata to be due to some evolutive troubles which depend on different toxic infections, and which may affect the developing embryo in all the stages of gestation.

As examples they mention congenital hydrocephalus and spina bifida, which are important signs of degeneracy, and are the result of an inflammatory lesion of toxic origin evolved during intra-uterine life.

R. G. Rows.

**ALBANY HOSPITAL.** First Report of Pavilion F, Department for (246) Mental Diseases, for the year ending February 28, 1903. J. M. MOSHER, *Albany Med. Ann.*, 1903, No. 4.

**ALBANY HOSPITAL** seems to have been the first general infirmary in the United States to open special wards for the treatment of

early cases of insanity. The experiment—for as such the establishment of this department has been openly regarded—has been watched with considerable interest throughout the country, and already there is a very general impression that it has been successful.

Dr J. M. Mosher, who has medical charge of the new wards, in his first annual report to the Board of Governors, states that 174 patients were admitted. Of these 57 were discharged recovered, 53 were improved, 43 were not improved and six died. Dr Mosher remarks that “the physicians of Albany had had long acquaintance with the suffering, incident upon the emergencies of mental diseases, and had been daily confronted with the disposition of patients whose needs exceeded the resources of the home. The inevitable result was recourse to the police and the jail with eventual commitment to an institution for the insane, even for transient disturbances, when temporary care would often have resulted in the restoration of health.” The medical and surgical staff of the Albany Hospital proposed that an additional pavilion should be constructed for mental cases. This proposal was adopted, and the wards were opened about the beginning of last year. “Following the notation of the Hospital wards, the new department was designated Pavilion F, thus happily escaping any distinctive or suggestive title, and bringing into prominence the fundamental fact of the modern conception of insanity, that it is a disease, and that its victims are entitled to the same consideration as the bodily sick or injured, and present a claim for treatment which a hospital should not ignore.” Dr Mosher further says: “It has been demonstrated, in this first year, that mental patients of all classes may be received upon voluntary request, and that a small minority resent the confinement and cannot be held. Of the patients who, from dissatisfaction with the surroundings, homesickness, or other cause, withdraw, several have returned to their homes benefited by their short stay, and others have finally needed the intervention of the law and formal declaration of insanity. The number of malcontents is not greater than in other departments of the Hospital.” He thinks that no definite rule can be established as to the length of time the patients should remain in hospital. He recognises that there are many cases for which treatment in these hospital wards is unsuitable. “It may be said in this connection, and as a result of anxious study of the possibilities of our Pavilion, that in certain probably curable cases we have failed to secure results. This is a demonstration of its limitations, from which it appears that patients whose condition does not demand the active medical treatment provided by a general hospital should not be too long detained, when so-called moral or mental methods, diversion by occupation or by well-ordered recrea-

tion, are necessary for distraction of ideas from a morbid to a normal channel. It appears consequently that we have done more than establish an emergency hospital for temporary care. Prospect of cure has been extended to many patients whose minds have been seriously involved. The principle may be stated that *any patient, whose case may be regarded as curable with the means at hand, should be offered the ministrations of this pavilion.*"

W. FORD ROBERTSON.

**THE GEOGRAPHICAL DISTRIBUTION OF LUNACY IN ENGLAND, SCOTLAND, AND IRELAND (URBS ET RUS). THE EFFECT OF MIGRATION AND THE DEATH-RATE UNDER FIVE.** J. F. SUTHERLAND, M.D., XIV. Int. Medical Congress, Madrid, 1903. Section de Maladies Mentales, &c.

THE geographical distribution of the lunacy of a country may most opportunely be considered as soon as possible after the enumeration or census has been taken by Government Departments. The statistics furnished in this way are the most reliable and complete, as they include those officially known to the Public Departments as well as those not.

Two years ago (1901) that enumeration was made for Great Britain and Ireland, with a population of 41 millions residing *urbe, oppido, et rure*, and engaged in pastoral, mining and industrial pursuits. It will be seen that the ratios of insane per 10,000 of population for England, Scotland and Ireland were respectively 40, 45 and 56. The maximum ratio for an English county was 63, the minimum 23; for a Scottish county, 84 and 29; and for an Irish, 96 and 30.

The considerable difference between the ratios for the three countries, and the still more remarkable differences between the extremes of each country, are suggestive. It may be asked why Ireland's lunacy should be 15 per cent. greater than Scotland's and 28 than England's. And the explanations which are forthcoming for these differences in the three countries as a whole will turn out to be true in a striking manner when the parts of each of the three countries are contrasted.

The distribution of lunacy in Great Britain and Ireland is somewhat analogous to what is found in most European countries, and perhaps attributable to the same causes, viz.: first, the emigration of a large proportion of the healthy manhood of European countries to North and South America and to Australia; and second, the migration of a still larger proportion of the manhood and womanhood from rural to urban areas in obedience to the

inexorable economic laws of our times. In this connection I may observe that the dolico-cephalic Teutonic and Iberian races have shown a preference for urban life. As a rule it is the fittest who migrate.

One result of the operation of these two forces, migration and emigration, is that the marriage-rate and, as a consequence, the birth-rate, are declining in the rural districts just as the population as a whole in such areas is either stationary or declining. Another result is that the weak and sickly products of the birth-rate, both mental and physical, are left behind, and that sexual selection is so restricted that the heredity phase of insanity is of necessity accentuated in rural areas.

Add to these two forces constantly in operation another and striking one, viz., the death-rate under five years of age, which in urban and industrial areas is double, treble, and even quadruple that of rural areas, and there is to hand ample explanation of ratios of insane to population as widely apart as 30 and 90 per 10,000 in the same country. This enormous infant mortality in urban areas, suggestive of a ghastly hecatomb to the conditions of modern life, has the effect of removing thousands of lives, which if they had not succumbed to urban insanitary environments, to neglect, and to the exanthematous diseases under present conditions incidental to and inseparable from city life, would have swelled the total and given much higher ratios for urban areas than are revealed by the recently compiled statistics.

Assuming as I have done the proposition that given large areas of a country whether urban or rural, and given large populations, the productivity of lunacy will not vary except within very narrow limits unless it can be shown that special agencies are at work in one and not in another. The acceptance of a proposition such as this, it will be admitted, is necessary.

No doubt such specially productive agencies exist and are at work, but they are at work, strangely enough, in localities where the lunacy ratio is found to be lowest. In urban areas such potent factors as alcohol and syphilis leading to general paralysis of the insane, dementia and mania, and the stress and strain of life are constantly and actively at work. In rural areas, on the other hand, there are, so far as I know, no counter-balancing productive forces, and consequently one would naturally look for much lower ratios there. It should be said that the only special cause in rural districts, and it is a cause not to be compared for a moment in potency with alcohol, syphilis, stress, etc., is the restriction of sexual selection following migration and emigration in stationary or declining populations.

But the statistics show quite the reverse of what one would expect, and that to a remarkable extent.

The explanation of these enormous ratio differences is to be found in the abnormally high child mortalities in urban areas compared with rural.

Thus in England in the Liverpool and Manchester urban areas the ratio of insanity is 35 per 10,000 of population, while the mortality ratio under 5 is 86; in the Birmingham urban area it is 34 and 73 respectively, and in the Newcastle urban area 26 and 85. But in a large rural area of England the figures are reversed, being 55 insane per 10,000 of population and 43 per 10,000 deaths under 5.

In Wales in the Cardiff urban area the insanity is 28, the mortality under 5 being 80, while in rural Wales the figures respectively are 32 and 46.

In Ireland in the Dublin and Belfast urban areas the lunacy ratio is 32, the death-rate under 5, 71; while in rural Ireland the figures are reversed, being 66 and 30 respectively.

In Scotland, with which I am more familiar, it will be found that in the Glasgow and Edinburgh urban areas the ratio of insanity per 10,000 is 35, while the death-rate under 5 is 85 per 10,000. Again in rural and insular Scotland (one half) the insanity-rate is 75, and the death-rate under 5 is only 21.

These figures call for reflection. At a glance it will be seen that where this mortality is high the lunacy ratio is low, and conversely where this mortality is low the lunacy ratio is high. Remarkable extremes are met with both in regard to lunacy ratios and mortalities under 5, but by levelling up or down the death-rates under 5, when comparing the lunacy of urban with rural areas, a very different complexion is given to the figures, and the extremes referred to approximate very considerably. This method of adjustment seems perfectly fair if anything like the truth is to be arrived at. If a population such as that of which I am treating has got in its midst 40 to 50 insane per 10,000 of population, and that the survival of the fit means such a sacrifice of infant life, it may be accepted without much disputation that the thousands under 5 years of age decimated annually in cities by exanthematous disease, by neglect, bad nursing, and injudicious dieting would have contributed as their quota of insane 60 to 70 per 10,000, for there is no gainsaying the fact that it is the imbecile as well as the physical weaklings of the birth-rate who most readily succumb to unfavourable environments. The law of "natural selection" may explain a good deal, but into that I cannot enter.

I cannot close this statement without referring to the widening portals to certification and to lunacy registers, and to institutions in every country in Europe. Seeing there is no scientific definition of insanity, as there is none of sanity, the widening *entrée* is not surprising, and as a consequence cases of degeneracy, senility,



eccentricity, vesania readily find their way to official registers. But there is an everyday workable definition of lunacy. The mere presence of hallucinations, delusions and illusions brings no one within the four corners of Acts of Parliament. It is only when these govern conduct, which after all is the safest test, in such a way as to lead to actions injurious to self, to the lieges, and to property, that the law intervenes, and gives its protection to all three.

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## Original Articles

### THE CENTRAL NERVOUS SYSTEM OF AN ANENCEPHALIC FŒTUS.

By DAVID WATERSTON, M.D., F.R.C.S.E.,

and

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THE pathology of the condition of anencephaly is not yet thoroughly understood, and it is not our intention to discuss the different theories of its production, but we wish to draw attention to the minute structure of the spinal cord in a case of this kind, in which the entire central nervous system above the level of the medulla oblongata was absent. Various degrees of anencephaly are found, varying from those extreme forms in which the nerve elements in the brain and spinal cord are absent, and only the posterior spinal ganglia are left, to slight forms, in which there is absence of part of the encephalon alone. Very few descriptions on similar lines have been published, and as the degree of anencephaly varies in different specimens, the condition of the nervous system also shows considerable variation.

Two cases of comparatively recent date have been published, but in one of them (1) the upper part of the spinal cord had been destroyed, and in the other (2) the mid-brain was present. In our specimen there was practically nothing beyond the spinal cord and the medulla, and therefore we anticipated that the sections of the cord would demonstrate merely the presence of the grey

matter and of the endogenous and ascending tracts, together with any alterations that might have resulted from the absence of tracts descending from higher levels.

In the case described here the embryo was a well-developed full-time male, showing all the characteristic features of anencephaly of moderate degree.

On naked eye examination, the central nervous system was represented by an entire spinal cord and a prolongation of the cord, 20 mm. long, of the nature of a cylindrical mass which passed through the foramen magnum, and ended in a free conical stump. The central canal opened on the dorsal surface of this mass 6 mm. from its upper end, and some nerve roots came off on either side.

The cord measured 12.3 cms. in length, and presented apparently normal cervical and lumbar enlargements.

The cranial vault was absent, and the cranial cavity was filled by a reddish brown vascular mass, adherent to the base of the skull, into which the central ends of some nerves passed. The optic nerves were short, and ended by blending with the fibrous tissue covering the body of the sphenoid. There was no chiasma.

*Method.*—The cord was divided into segments corresponding to the origin of the spinal nerves, and the axis within the cranial cavity was divided transversely into four small segments, and sections were cut and examined from each segment, after staining with toluidin blue and by the Weigert-Pal method.

*Sacral region.*—In the sacral region there was no marked alteration from the condition normally found in a well-developed foetus, beyond the fact that the lateral columns of the white matter appeared to be rather small, but the grey matter was everywhere separated from the surface of the cord.

The motor cells of the anterior horns were large, numerous, and well-developed—as many as 70 to 80 in one section on one side, and the central canal appeared rather large.

The ciliated epithelium was deficient on the posterior wall of the canal. (Fig. 1.)

*Lumbar region.*—In the region of the lumbar enlargement the grey matter was well formed, and there was a large substantia gelatinosa, into which medullated fibres entered.

The motor cell groups were distinct, and were made up of large, highly granular motor cells.





FIG. 1.—First sacral segment ( $\times 20$ ).



FIG. 2.—Second lumbar segment ( $\times 20$ ).



FIG. 3.—Mid-dorsal segment ( $\times 20$ ).



The central canal was well formed, but contained some red blood cells.

The lateral columns of the white matter were small, while the posterior columns were large, well medullated, and showed no subdivision into root zones. (Fig. 2.)

*Dorsal region.*—The structure of the cord in this region showed some distinct alterations. (Fig. 3.)

The difference in relative size between the antero-lateral and the posterior columns was now very distinct, and the lateral columns were fissured on the surface, especially in the region of the direct cerebellar tract, which was thrown into folds.

The direct cerebellar tract was first seen at D 10, and was distinct above this level, and at D 9 there was an indication of a morbid change, more obvious higher up, in the presence of masses of red blood cells in the interior of the central canal, and in the nerve tissue.

The posterior columns were well myelinated, and there was no distinction between the postero-median and postero-external tracts.

In the grey matter there was a distinct intermedio-lateral horn, with numerous nerve cells. (This was absent in Bulloch's case.)

Some doubt has been thrown upon the existence of Clarke's column in such cases, but we were able to find it in our specimen, composed of rounded cells, which stained faintly. The Nissl bodies had either undergone degeneration, or were imperfectly developed.

In and above the mid-dorsal region, the whole substance of the cord was studded with numerous patches, which consisted of red blood cells lying in masses in spaces in the grey and white matter, or scattered at intervals among loose tissue.

The distribution of these patches was irregular, but they became more numerous higher up, and they formed a curious feature, which must have some connection with the pathology of the condition.

With low power magnification the appearance was very similar to that shown by Purves Stewart from a case of acute myelitis in the last number of this Journal, but in our specimen the aggregations were composed almost entirely of red blood cells.

*Cervical region (C 7). (Fig. 4.)*

*Grey matter.*—The motor nerve cells were numerous, well formed, and were arranged in distinct groups, and the anterior horns contain numerous medullated fibres. There was a large cap of substantia gelatinosa over the apex of the posterior horns.

*White matter.*—There were two deep fissures in the margin of the antero-lateral columns, while the direct cerebellar tract was distinct, and thrown into folds by the fissures on the surface. There was a pale non-medullated area corresponding to the tract of Lissauer, while the direct and crossed pyramidal tracts were absent, as there was no area of non-medullated fibres to mark their usual position, as should be the case in a normal cord.

In the lower cervical region, there was a septum between the tracts of Goll and Burdach, though both were equally well medullated, but, at a higher level, there was some difference, the postero-internal tract being uniformly paler in Weigert-Pal specimens.

At the fifth cervical segment, there were two central canals, and, at the third, there was one large well-formed canal, and beside it a second rudimentary one, with ependymal cells scattered throughout the substance of the grey commissure.

The appearances remained similar up to the level of the second cervical segment, in which emerging medullated fibres of the spinal accessory nerve could be seen; but above this level, in one of the lower segments of the intra-cranial mass, there were three distinct, well-formed central canals.

Examination of the segments of the mass at different levels showed it to be made up of masses of grey and white matter arranged in a complex fashion, and with numerous dilated spaces containing well-formed red blood cells.

Two chief points of interest may be noted. (Figs. 5 and 6.)

The ventral surface of the mass was clothed by a narrow uniform mass of apparently embryonic nerve tissue, and, in the interior of this, was the tissue representing the lower part of the medulla oblongata.

Further, emerging nerve fibres could be seen in the position of the 12th cranial nerve, springing from a well-formed group of cells, but the 11th and 12th cranial nerves appear to be the only ones whose nuclei are represented.

*Conclusions.*—The principal features of the cord are the following:—

As was anticipated, the posterior columns are well formed,

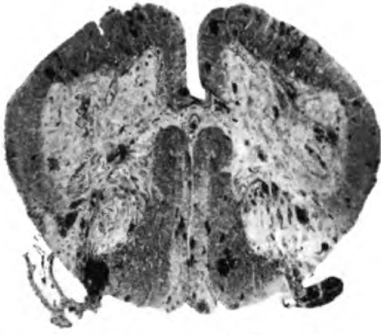


FIG. 4.—Sixth cervical segment ( $\times 10$ ).

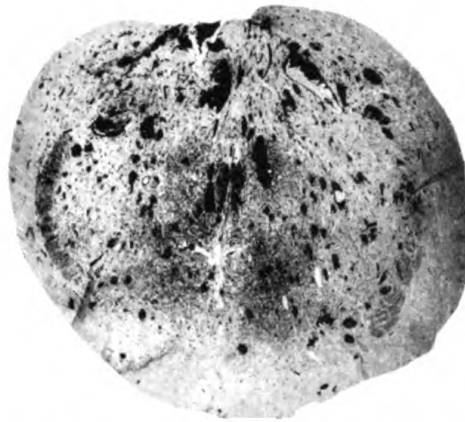


FIG. 6.—Section through upper end of intra-cranial mass ( $\times 10$ ).



FIG. 5.—Section through mass just above spinal cord ( $\times 20$ ). Three "central canals" can be seen (*a*).



and of the usual size and shape. Absence of descending fibres does not therefore have any appreciable effect in diminishing these columns.

The lateral columns are the most affected, but the least change is seen in them in the region of the enlargements.

In the thoracic region, the surface of the lateral columns is fissured in a manner similar to that noticed in absence of a curved pyramidal tract.

The absence of fissuring in the lumbar and cervical regions is probably due to the presence of a proportionately large number of short endogenous commissural fibres, which make up a great part of the white matter in those regions.

The anterior columns are affected only to a moderate extent.

The grey matter is well formed, and contains normal motor cells, and the intermedio-lateral horn is present in the dorsal region.

The vesicular column of Clarke contains cells which resemble cells that have undergone chromatolysis, and this fact may be associated with the absence of a terminal organ for their axis cylinders in the cerebellum.

The central canal as we pass upwards becomes first duplicated and eventually triplicated in some places. (Fig. 5.)

Duplication of the central canal has hitherto been recognised only in (1) duplication of the whole cord, (2) duplication of the whole or part of the grey matter, and (3) obliteration of the central canal, and it is difficult to account for the condition in our specimen, unless it be due to a filling up of the primitive central canal by blood cells.

The cord in its upper half is remarkable for the presence of numerous blood-vessels of large size, and of masses of red blood cells, resembling hæmorrhages in both the grey and white matter.

In Bulloch's case the white matter was small in proportion to the grey, the anterior horn contained normal multipolar ganglion cells, Clarke's column contained very few cells, and the lateral horn of the dorsal region was quite rudimentary. In the white matter, the pyramidal, direct cerebellar tracts and the tract of Lissauer were absent.

Our observations, therefore, show a condition different in several respects from that noted by this observer.

Vaschide and Vurpas noted also the existence of dilated blood spaces, filled with blood corpuscles, both red and white. In their

specimen there was the same "degeneration" of fibres in the postero-internal tract in the upper cervical region alone, but they describe a degenerated condition of the nerve cells in the anterior horns such as was nowhere detected in our specimen.

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Vaschide et Vurpas, *Nouvelle Iconographie de la Salpêtrière*, 1901, No. 5.
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### **HISTOLOGICAL EVIDENCE OF THE PRESENCE OF AN ORGANISM RESEMBLING THE KLEBS-LÖFFLER BACILLUS IN CASES OF GENERAL PARALYSIS OF THE INSANE.**

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IN a paper recently published by Dr G. Douglas M'Rae, Dr John Jeffrey and myself (1), there were recorded the results of some bacteriological investigations into the pathology of general paralysis of the insane which seemed to indicate that in this disease there is constantly an infection of the alimentary and respiratory tracts by an organism indistinguishable from the Klebs-Löffler bacillus. Grounds were stated for believing that this bacillus exercises a pathogenic action in general paralysis, and the hypothesis was enunciated that the disease is the result of a chronic toxic infection from the respiratory and alimentary tracts, permitted by general and local impairment of the defences against bacteria, and dependent upon the excessive development of various bacterial forms, but especially upon the abundant growth of a Klebs-Löffler bacillus of modified virulence, which gives the disease its special paralytic character.

At the present moment this hypothesis rests merely upon a certain amount of presumptive evidence which, though its exact weight may be a matter of considerable difference of opinion, certainly cannot be explained away by any existing data. Before it can be regarded as refuted or established, additional evidence must be collected from many sides, the most important being that to be obtained from experimental investigation into the pathogenic action of the bacillus. On the threshold of the



inquiry a question naturally arises which, if it cannot be answered satisfactorily, must throw grave doubt upon the validity of the hypothesis. It may be asked, What ground is there for believing that this supposed Klebs-Löffler bacillus that can be isolated from the alimentary and respiratory tracts of cases of general paralysis has any more significance in this disease than the occasional presence of a similar organism in various morbid conditions that are not diphtheria and even in well persons? Probably only the experimental evidence can give the final answer, but apart from such testimony there are some facts which strongly favour the view that the organism is exercising a pathogenic action in cases of general paralysis. Two of these facts have been established by the investigations of which an account has already been published. They are that the bacillus was ascertained to be present in the alimentary or respiratory tract of all of the twenty cases investigated post-mortem, and that in a considerable proportion of cases the organism was obtained from the brain, which it had probably reached (the evidence does not as yet warrant any other conclusion) in consequence of a terminal general infection. A third fact of no less weight is that histological investigation of a series of cases of general paralysis not only confirms the conclusion that this bacillus is constantly present either in the alimentary or respiratory tract, or in both, but proves that in a large proportion of cases it is there in very great numbers, being indeed in some instances the predominating organism. The purpose of this paper is simply to give a brief record of the observations upon which this statement is based. The evidence that can be shown to exist of excessive development of various bacterial forms in the alimentary canal, of local infection in various situations, and of the occurrence of chronic inflammatory and atrophic changes in the walls of the alimentary and respiratory tracts, I hope to deal with at another time. I may state now, however, that as regards the morbid alterations in the alimentary tract, the histological study of a series of forty cases of general paralysis has only served to establish more firmly the conclusions formulated two years ago (2). In my experience chronic catarrhal changes of a severe nature are constant in this disease, either in the stomach or small intestines, generally in both.

Histological evidence of the presence of an organism resem-

bling that isolated by bacteriological methods has been sought for in twenty cases. Thirteen of these are included in the series that formed the subject of the bacteriological research already recorded. The alimentary tract has been examined in all of the cases. The respiratory tract has, however, been studied in only five, for the simple reason that it was not until comparatively recently that the possible importance of chronic bronchial infection was realised and steps taken to obtain the tissues necessary for investigation of the subject. For the same reason the tonsils have been examined in only a few of the cases.

Before detailing the results of the enquiry, it is necessary that I should allude to some of the difficulties that lie in the way of the identification of this organism resembling the Klebs-Löffler bacillus in sections, and to the staining methods that I have employed. It is well known that the identification of the diphtheria bacillus in films, whether of material taken directly from the throat or of cultures, is often a matter of considerable difficulty and uncertainty. Some bacteriologists are satisfied with the evidence obtainable by the use of Neisser's method, whilst others declare that the reaction is not absolutely distinctive, and think that the method has no advantages over various other staining processes. Some would even go so far as to say that the only conclusive evidence is that to be derived from the experimental inoculation of a pure culture into a susceptible animal. If the identification of the Klebs-Löffler bacillus in films is often difficult, in sections it is very much more so. Indeed, it is probably within the mark to say that few, if any, bacteriologists would at the present day consider themselves competent to give a definite opinion that a bacillus seen in a histological preparation is a Klebs-Löffler bacillus, especially if it occurs apart from a typical diphtheritic exudation. Neisser's method, upon the evidence of which very many rely in the examination of films, is not suitable for sections. Under such circumstances one might almost be justified in abandoning as futile an attempt to gain any definite information regarding the presence or absence of this organism by histological methods. In this investigation, however, the question that had to be decided was not that of whether the Klebs-Löffler bacillus is present in sections of certain organs, but simply whether a bacillus morphologically identical with that which has been isolated by bacterio-

logical methods is commonly demonstrable. The question of the identity of this organism with the Klebs-Löffler bacillus must for the present remain a moot point. As yet it can only be affirmed that its cultural and morphological characters resemble those of the diphtheria bacillus.

Using pure cultures of the bacillus isolated from cases of general paralysis, I have endeavoured to devise some distinctive method of coloration that might be applicable to sections. These experiments have so far been without success, excepting that they have led to an adaption of Neisser's method which has proved of some service. The details of this process, which is carried out upon sections fixed upon the slide, are as follows. The sections are stained for several hours, or over night, in Neisser's methylene blue solution of five times the normal strength as regards the methylene blue and alcohol. The composition of the stain is therefore as follows:—Methylene blue (Grübler) 5 gm., 96 per cent. alcohol 100 c.c., distilled water 950 c.c., glacial acetic acid 50 c.c. This fluid must be carefully filtered before use. The sections after being washed in water are counterstained for two minutes in Neisser's Bismarck brown solution (2 grms. of the dye to 1 litre of water). They are then washed in two or three changes of water. The colours are next fixed in the tissues by means of a 5 per cent. solution of ammonium molybdate, which should be allowed to act for two or three minutes. This reagent is then carefully washed out in water, and the section is dehydrated with absolute alcohol cleared in turpentine-xylol and mounted in xylol-balsam. All the tissues that I have stained by this method have been fixed in 5 per cent. formalin. It is doubtful if there is any advantage in using the counterstain. It certainly tends to obscure some of the bacilli. The methylene blue stain alone, fixed by means of ammonium molybdate, gives an equally distinctive reaction, the bodies of the bacilli staining of a pale blue tint, and the metachromatic granules, when present, appearing as clearly defined purplish points. It is absolutely necessary for the success of this method that the tissues should have been secured in a fairly fresh state.

It is now well known that the Klebs-Löffler bacillus, from which the bacillus found in cases of general paralysis cannot be ascertained to differ in any essential respect, has very varied

morphological characters, and that the type with prominent metachromatic granules is only one of many. Wesbrook (3) has divided the numerous varieties that may be observed into three main groups, namely, granular, barred, and solid-colour forms. The granular forms, though including those that show metachromatic granules, do not necessarily exhibit this feature. These considerations justify the conclusions that it is at most only a small proportion of the organisms resembling the Klebs-Löffler bacillus that are capable of being distinctively stained by this methylene blue method, and that when a few with metachromatic granules are observable in a preparation they are indicative of the presence of many more bacilli that are not clearly revealed. This opinion is confirmed by the study of the isolated groups of these organisms that may occasionally be seen in such preparations.

Another method that I have relied upon as an aid in the identification of the bacillus in sections is Weigert's modification of Gram's method. When one has become familiar with the appearances of the bacillus in film preparations of pure cultures stained by this method, it is generally possible to recognise the same organism in its granular and barred forms when it is present in sections similarly stained.

Using these and other methods, I have been able, as already indicated, to recognise in the catarrhal exudations in the alimentary or respiratory tracts of all of the twenty cases investigated, a bacillus identical in form and staining reactions with the organism isolated by culture methods. In most of the cases the bacilli were to be observed in considerable numbers. In eight of the cases they were present in very great numbers. In those eight cases their ascertained distribution was as follows:—In the stomach in three cases; in the stomach and lower part of the ileum in one case; in the crypts and at the surface of the tonsils in two cases; and in the bronchi and stomach in two cases. The organism was found in the respiratory tract in each of the five cases in which this region was examined. An idea of the enormous numbers in which the bacilli are sometimes present may be gathered from Fig 1. In addition to being present in catarrhal exudations, the bacillus was frequently to be observed in the ducts of the gastric glands, in the ducts of exhausted compound mucous glands in the respiratory tract, and occasionally

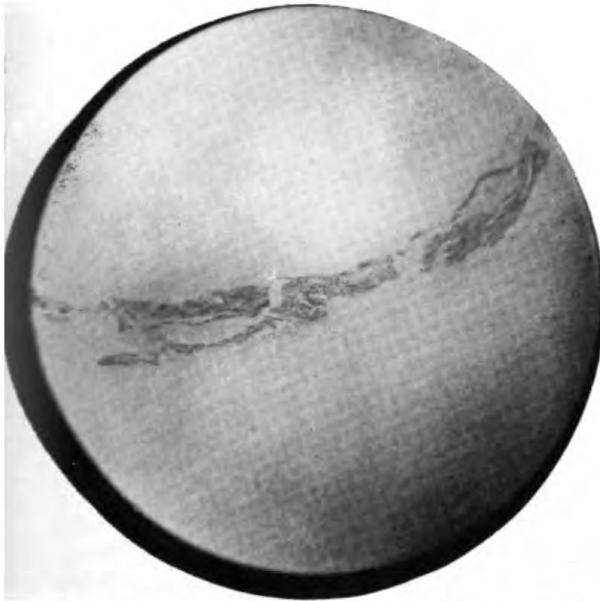


FIG. 1.—Transverse section of trachea from a case of general paralysis. Stained by Gram-Weigert method. Magnified about 10 diameters. The dark band is lying on the surface and is formed almost exclusively of organisms resembling the Klebs-Löffler bacillus.



FIG. 2.—Transverse section of ileum from a case of general paralysis. Hæmatoxylin and eosine ( $\times 100$ ) shows inflammatory changes. The exudation on the surface contained very numerous bacilli resembling the Klebs-Löffler bacillus.

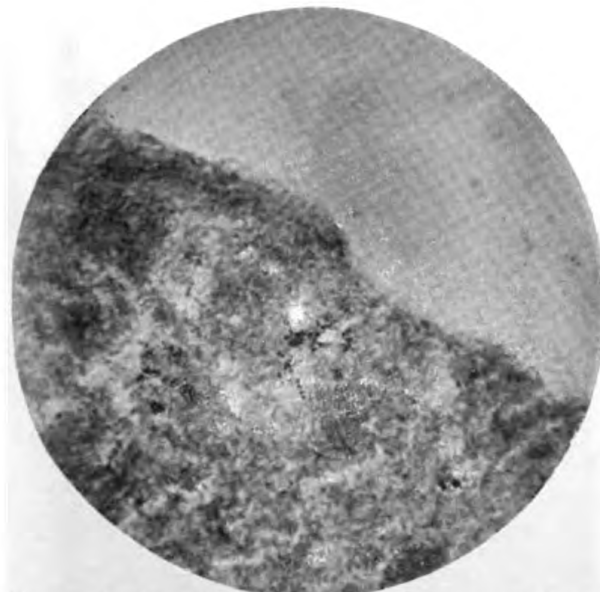


FIG. 3.—Portion of inflammatory exudation on surface of ileum shown in Fig. 2. Stained by adaptation of Neisser's method to sections ( $\times 1000$ ). Shows group of bacilli with metachromatic granules.

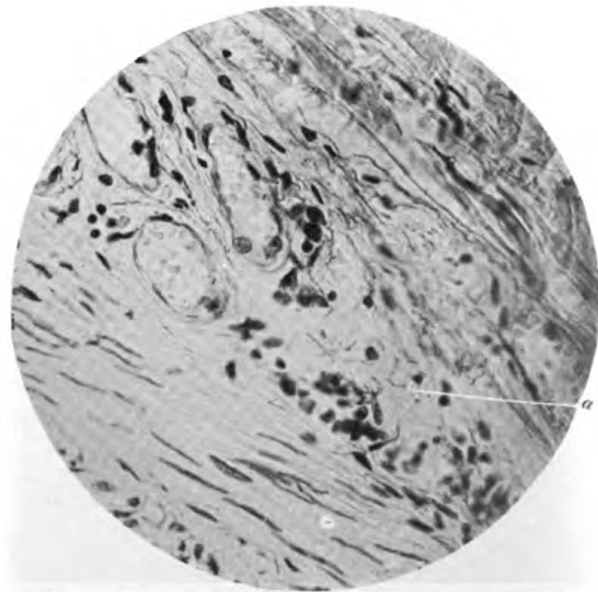


FIG. 4.—Section of bronchus from a case of general paralysis. Carbol thionin ( $\times 600$ ). *a*. Filamentous organisms lying in lymphatic spaces in sub-mucosa.



also in the superficial layers of an inflamed tonsil or gastric mucosa.

The fact that this special bacillus was present in these eight cases in very large numbers cannot be accounted for by any post-mortem development, as the organism does not grow at the ordinary temperature. These organisms must therefore represent a part of the bacterial flora of the contents of the alimentary or respiratory tract during the 24 or 48 hours that preceded death. In what respects this flora essentially differs from that at an earlier period, is a question upon which the investigation here recorded naturally throws no light.

Although I do not intend in this paper to record the more recent observations upon which are founded the conclusion that the general paralytic is the subject of a mixed toxic infection from the alimentary and respiratory tracts, there is one other infective phenomenon that has been revealed in the course of the study of these twenty cases which it is necessary to allude to here. I refer to the presence of a filamentous organism in the lymphatics of the walls of the alimentary or respiratory tract. This organism has been observed in enormous numbers in five out of the twenty cases investigated, namely, in two cases in the stomach wall alone, once in the stomach and ileum, once in the ileum alone, and once throughout the whole extent of the bronchi and trachea. In the last instance it spreads through the lymphatics of the lungs and has reached the bronchial glands.

This organism appears as smooth threads of various lengths and, as seen in carbol thionin preparations, shows alternate pale and dark portions, the latter being the shorter. Bacillary forms are common. They generally show a lightly stained central portion and at each rounded end a comparatively dark polar granule. They are about the size of the largest forms of the Klebs-Löffler bacillus. The individual segments of the thread forms are usually somewhat longer and also broader. The organism is very clearly defined by deep carbol thionin staining and differentiation with absolute alcohol. (See Fig. 4.) It is also fairly well stained by the methylene blue method described. It does not retain the colour in preparations by Gram's method, but to this rule there are some exceptions. It is not acid-fast after staining with carbol fuchsine. The organism was morphologically identical in the five cases. It was also

observed in the wall of the stomach of one of the control cases examined, a woman who had suffered from recurrent mania and who died at an advanced age. The exact nature of the final illness, which lasted for two or three months, was not determined.

When I first saw this organism in the wall of the stomach of a case of general paralysis, there seemed to me to be no reason for regarding it as representative of anything more than an accidental terminal infection. There was certainly no ground for suspecting it to be an important etiological factor in the disease. In the further course of the investigation, however, a series of facts have come to light which suggest that it is the streptothrix or actinomycotic form of the organism resembling the Klebs-Löffler bacillus. These facts are briefly as follows. The thread form has been ascertained to be present in the walls of the respiratory or alimentary tract in one-fourth of the cases investigated, and there are good grounds for supposing that it may have been missed in some of the others in which the respiratory tract was not examined. The bacillary form of the organism is morphologically identical with some of the granular and barred forms of the diphtheroid organism, excepting that it tends to be rather larger. Films made from old (four to eight days) byno-hæmoglobin agar cultures of the latter organism, and stained deeply with carbol thionin, show forms that are indistinguishable from the shorter of the threads seen in the tissues. I have been unable to observe any long threads in cultures, but have seen many that extend to four segments. In some Gram preparations of tissues containing the filamentous organism, both its bacillary and thread forms may in a few instances retain the stain, and they have then morphological characters essentially the same as those of some of the diphtheroid bacilli. In sections coloured by the methylene blue method, the filaments show staining affinities similar to those of certain forms of the supposed Klebs-Löffler bacillus. Although meta-chromatic granules are not as a rule visible, I have a set of preparations in which the organism clearly exhibits them.

Stronger evidence than is afforded by the above facts is needed to warrant the conclusion that these two forms are morphological varieties of the same organism. The point is however, one that it is of some moment to decide, for although proof that the two forms are in no way related would not





weaken the case in support of the hypothesis enunciated by my colleagues and myself, the establishment of their identity would greatly strengthen it.

I have endeavoured to ascertain if the diphtheroid organism and the thread form are demonstrable in sections of the brain in cases of general paralysis, but the results have so far been entirely negative. Various staining methods reveal abundant faintly coloured particles and even short threads, especially in the walls of inflamed vessels; but there is as yet, I think, no warrant for regarding them as anything but the granular matter derived from disintegrating tissues, or formed in consequence of disordered local metabolism. I have not been able to find any organisms resembling the bacillus described by Piccinino (4), although I have not used his special technique.

It would be premature to discuss the bearing of these observations upon the question of the pathogenesis of general 'paralysis. It is only claimed that the results are in harmony with the hypothesis that has already been advanced on the ground of bacteriological researches. The pathogenic action of the bacillus we have isolated can only be determined by experiment, and when the evidence derived from this line of investigation has been brought forward, I hope to deal with the problem of pathogenesis, as affected by these and other recently recorded observations.

I have to acknowledge my indebtedness to Dr M'Rae, Dr Jeffrey, Dr David Orr, and Dr Neil T. Kerr for the tissues used in this investigation.

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\* These references were inadvertently omitted in the paper published in the May number of the *Review*, and are therefore added here.

## Abstracts

### ANATOMY.

**A CONTRIBUTION TO THE STUDY OF LOCALISATION IN THE  
(248) HYPOGLOSSUS NUCLEUS.** C. PARHON et CONSTANCE  
PARHON, *Rev. Neurol.*, 1903, p. 461.

THE nucleus hypoglossus is divisible into several groups of cells : a ventral, which also lies median, a dorsal, a lateral, present only in the middle and upper third of the nucleus, and towards the cerebral end another small group between this latter and the dorsal.

In the present paper the authors describe the changes found in the nucleus hypoglossus in a case in which a carcinoma had destroyed the greater part of the M. palatoglossus, pharyngoglossus and amygdaloglossus and had involved the posterior part of the styloglossus and of the superior lingual, but had spared the genioglossus, hyoglossus and transverse and inferior linguals. Typical secondary reaction was found in the cells of the lateral and ventral groups, from which it may be concluded that these are the groups concerned in the motor innervation of the affected muscles.

As in a previously published case of similar nature the lateral group of cells was normal while the growth did not involve the M. pharyngoglossus, palatoglossus and amygdaloglossus, it appears that these are the muscles innervated from this group.

GORDON HOLMES.

**SECONDARY DEGENERATION FOLLOWING UNILATERAL  
(249) LESION OF THE CENTRAL MOTOR CORTEX.** SUTHER-  
LAND SIMPSON, *Internat. Monatsschr. f. Anat. u. Physiol.*, 1902.

THE author performed a series of experiments upon cats, in which the Rolandic area, as determined by faradisation, was "under-cut" on the left side in such a way as to sever the long descending fibres without injuring the basal ganglia ; the animals were tested for motor and sensory disturbances on the second and succeeding days, and after a few weeks were killed ; the central nervous system was then examined by the Marchi method. One dog and two monkeys were similarly treated. In testing for tactile sensation, a needle attached to a long stick was gently stroked over the limb whilst the animal's attention was distracted ; needle-pricks instead of the clip-test were used to test for pain ; and heat and

cold were tested by immersing the paw in water at 57° and at 0° respectively.

*Symptoms.*—In every case the lesion was followed by motor paralysis of the opposite limb. This was quickly recovered from as regards the “associated movements” of walking and running, but ability to perform isolated (voluntary) movements was often not restored before the animals were killed.

The sensory changes were less uniform. In half the cases tactile sensibility was absent at first, in some it was delayed but present, in others it was unaltered, and in one case definite hyperæsthesia was present on the paralysed side. These symptoms usually passed off in three or four days. Heat did not appear to be appreciated as such on the affected side, but seemed after a short delay to cause pain more acute on this side than on the other. Cold was appreciated in eight out of thirteen cases.

*Degenerations.*—In two cases parts of the hemispheres and the thalamencephalon were examined; in all the cases series of sections were prepared from the mesencephalon to the sacral cord. The following degenerated fibres were found:—

1. Fibres passing from the internal capsule to the optic thalamus in the posterior part of which many fine fibres were present.
2. Fibres passing through the corpus callosum to the right centrum semi-ovale, but none to the right internal capsule.
3. From the anterior part of the internal capsule to the grey matter of the subthalamic region of the same side.
4. Large numbers of fibres passing from the crusta to the anterior corpus quadrigeminum of the same side, and some to that of the opposite side.
5. Fine fibres to the substantia nigra, nuclei pontis, and to the lateral portion of the mesial fillet.
6. Fibres passing to the reticular formation and to the neighbourhood of the substantia gelatinosa of Rolando in the medulla.
7. Crossed lateral and homolateral pyramidal fibres in the cord.

The author thinks that none of the degenerate fibres pass to any of the cranial motor nuclei or to the ventral horns of the spinal cord. He suggests that the fibres seen passing in the medulla to the formatio reticularis of the same and of the opposite sides may belong to the spinal motor decussation, and are destined for the lateral columns of the cord, and that they may correspond to what has been described as Pick's bundle in the human subject. It seems difficult, however, to reconcile this view with the direction and course taken by the fibres of Pick's bundle in man, which, as I pointed out (*Brain*, 1901), strongly suggests a bundle which is ascending to its distribution.

STANLEY BARNES.

**THE PYRAMIDAL BUNDLE IN INFANTILE HEMIPLEGIA—**  
(250) **COMPENSATORY HYPERTROPHY OF THE PYRAMIDAL**  
**TRACT.** PIERRE MARIE et GEORGES GUILLAIN, *Rev. Neurol.*,  
mars 31, 1903, p. 293.

IN this paper is recorded a detailed account of the clinical history and pathological appearances found in the central nervous system of a boy with hemiplegia.

The child had convulsions at two years of age, which increased in frequency, and a month afterwards it was observed that he was partially paralysed on the right side. The child grew up, was able to go to school at intervals, and afterwards was able to work.

On physical examination the condition of the boy—which was of great importance in view of what was found at the post-mortem—was briefly this:—The right leg and right arm were atrophied when compared with the left, but at the same time their movements were not much restricted. He walked with ease, with only some slight dragging of the right leg, and with the arm and leg he could accomplish all the movements at the different articulations. His power of resistance was good. Sensibility was normal, and there was no aphasia.

On post-mortem examination, the pia mater covering the left hemisphere was a little difficult to detach, but not adherent to the surface.

The cortical lesion was a very extensive one, involving the whole of the outer surface of the left hemisphere with the exception of the anterior extremity of the frontal lobe and the occipital pole. On the mesial aspect of the hemisphere, the paracentral lobe close to the supramesial border was affected. There was also some atrophy of the right lobe of the cerebellum.

Microscopically in sections cut from the affected hemisphere a large number of the cells of the grey matter were destroyed. Some were found in each section, but separated from one another by wide intervals. Many of the fibres from the cortex were demyelinated, a great number had been destroyed, and an irregular sclerosis of the neuroglia was present.

When compared with the opposite side, the anterior extremity, the genu and the posterior extremity of the internal capsule were extremely atrophied. So in the crus and pons, the motor fibres from the left hemisphere were similarly involved.

On the right side the corresponding motor tracts were hypertrophied when compared with a normal brain. This atrophy of the left and hypertrophy of the right motor tracts was even more marked in the medulla, the left showing merely as a thin strip on the surface.

In the cord the right lateral columns of white matter were

atrophied, with an extensive sclerosis in the region of the crossed pyramidal tract. The atrophic condition on the lateral aspect of the right side could be traced to the lower end of the cord.

The right direct pyramidal tract was markedly hypertrophied, and was traced down to the lumbar region.

The authors desire to draw particular attention to the hypertrophy of the motor fibres of the right side in the medulla and cord. They regard the hypertrophy as compensatory, and mention cases where a similar condition has been observed.

Looking to the very free movement and considerable power which remained in the affected leg and arm, the case, they think, is confirmatory of their opinion previously formed that the pyramidal bundle is not indispensable for the transmission of voluntary movements. Other tracts exist in the cerebrum and medulla through which motion is possible, and at the same time the fibres of the hypertrophied pyramid assist in maintaining the functions of the paralysed side.

EDWIN MATTHEW.

**A DESCRIPTION OF SOME ANOMALIES IN NERVES ARISING (251) FROM THE LUMBAR PLEXUS OF A FŒTUS, AND OF THE BILAMINAR MUSCULUS PECTINEUS FOUND IN THE SAME FŒTUS: WITH A STUDY OF THE VARIATIONS AND RELATION TO NERVE SUPPLY IN MAN AND SOME OTHER MAMMALS.** EDWARD B. JAMIESON, *Journ. Anat. and Physiol.*, 1903, vol. xxxvii. p. 266.

In the first part of the paper a number of irregularities in the nerves of the Lumbar plexuses are described, especially a pair of Accessory Obturator nerves which have an unusually extensive distribution. Previous descriptions of like irregularities are appended.

In the second part a bilaminar Pectineus is described with its relation to nerve supply in this particular case. A classified account is given of the variations in the pectineus which have been formerly described as occurring in man, and of the constitution of the Pectineus in various other mammals. The nerve supply commonly met with in man and in other mammals is described, and the variations in nerve supply in man.

In conclusion an endeavour has been made to show that the Pectineus as now found in man and most mammals is only one part of what was once a double muscle, and that the variations are attempted retrogressions towards the primitive double type. The opposite conclusion is also suggested: that the muscle is progressing towards the assumption of a double or compound character.

AUTHOR'S ABSTRACT.

**ON THE DEVELOPMENT AND HOMOLOGUE OF THE MAM-**  
(252) **MALIAN CEREBELLAR FISSURES.** O. CHARNOCK BRADLEY,  
*Journ. Anat. and Phys.*, vol. xxxvii., 1903, p. 112 and p. 221.

IN this paper the writer deprecates the practice, so commonly followed by comparative anatomists, of taking the human cerebellum as the type with which to compare the cerebella of other mammals. The human brain is so highly specialised that it is futile to expect to find representatives of all the lobes and lobules of the cerebellum of man in the lower, and more especially in the smaller, mammalia. For this reason a simple type was sought for and the more complex forms compared with it. If the cerebellum of a small mammal (the Shrew or one of the lesser Bats, for example) be examined, it is found that there are only four fissures running across the vermis. For the sake of convenience, and from a desire to use no terms which would call up a preconceived idea, these fissures are named provisionally I., II., III. and IV., the enumeration commencing anteriorly. On account of its surpassing depth, fissure II. is always easily distinguished in any brain. It is further of importance because of its early appearance in the embryo.

For the same reason that the fissures are named numerically, the lobes separated by them are simply referred to as A, B, C, D and E. In cerebella in which the surface anatomy is more complicated, additional fissures make their appearance. In this way lobe A becomes divided into two lobules (A1, that nearer the anterior medullary velum, and A2) by a fissure called for the time being *c*. Lobe B is not complicated by any fissures of moment; but lobe C, in all but the simplest forms, consists of three parts—lobules C1, C2 and C3—between which are fissures *b* and *a*. Lobe D is also divided into lobules D1 and D2 by a fissure *d*. Lobe E is usually small, and never becomes subdivided.

That part of the cerebellum which lies lateral to the hemisphere, and which is not infrequently spoken of as the "flocculus," has been shown by Stroud to represent more than the flocculus of man. The homologue of the human flocculus is always comparatively small in the average mammal, and lies ventral to, and often almost hidden by, a more conspicuous lobule, for which Stroud suggested the name of *parafocculus*. In the paper we are abstracting it is asserted that the flocculus and parafocculus are not only independent structures, but are connected with different parts of the cerebellum, and have a different developmental history. The parafocculus is really an outlying part of lobe D; whereas the flocculus has a similar relationship to lobe E.

In the research with which this communication deals, both Comparative Anatomy and Embryology have been put under

tribute. Adult cerebella from members of most of the orders of mammalia have been examined, and the development of the fissures in the rabbit and pig has been investigated.

In order that the writer's conclusions may be more readily followed, the accompanying table, showing the parts in the human brain corresponding to the various divisions of the mammalian cerebellum, is given :—

FISSURES.	LOBES.		
	Lobus centralis	A1	} A.
c. Sulcus postcentralis		A2	
I. (Not named by Schäfer)	Lobus culminis		} B.
II. Sulcus preclivalis			
	Lobus clivi	C1	} C.
b. Sulcus postclivalis	Lobus cacuminis	C2	
a. Sulcus horizontalis magnus	Lobus tuberis	C3	
III. Sulcus postpyramidalis	Lobus pyramidis	D1	} D.
d. Sulcus prepyramidalis	Lobus uvulæ	D2	
IV. Sulcus postnodularis	Lobus noduli		} E.

#### AUTHOR'S ABSTRACT.

**THE "HIRNSTAMM" OF THE DOLPHIN** (*Delphinus delphis*.) Von (253) RUD. HATSCHEK und SCHLESINGER, *Arb. aus dem Neurolog. Institute an der Wiener Universität*, H. ix., 1902, S. 1-117.

FOLLOWING upon his description previously of the spinal cord of the dolphin, H. now gives, in collaboration, an account of the mid-brain, medulla and pons in the same animal. The examination was effected by the study of coronal sections made in series, representative sections from different levels being figured.

The authors confirm the absence of an olfactory apparatus in this animal, and in association the fornix is feebly developed, as also the commissura hippocampus and the septum lucidum.

The authors give a careful description of the size and disposition of the nuclei of the cranial nerves.

In connection with the nuclei of the third fourth, and fifth nerves, the large size of the orbicularis palpebrarum muscle in

cetaceæ is pointed out, and, apparently in association with the great development of this muscle, a large (6 mm. in dorso-ventral direction) elliptical nucleus situated in the dorsal part of the oculo-motor nucleus.

The description of the other nuclei and of the tracts in the regions examined is careful, and the work is one which should be consulted by every writer on the comparative anatomy of this part of the nervous system.

DAVID WATERSTON.

### PHYSIOLOGY.

**CO-ORDINATING PATHS IN THE POSTERIOR LONGITUDINAL (254) BUNDLE.** E. H. FRASER, *Proc. Scott. Micros. Soc.*, vol. iii., p. 258.

THE material for this paper consisted of the central nervous systems of cats and monkeys, prepared by Marchi's method. Four groups of cases could be described according to the position of the lesion established:—

Group  $\alpha$  consisted of those animals in which the posterior longitudinal bundle was cut in its course beneath the lower part of the floor of the fourth ventricle.

Group  $\beta$  was formed by those animals in which the bundle was cut at a slightly higher level in the pons. (Both bundles were involved in the lesion in most of these cases.)

In group  $\gamma$  Deiters' nucleus was destroyed on the right side.

In group  $\delta$  the sixth nucleus was destroyed on the right side by a lesion which did not directly injure either Deiters' nucleus or the posterior longitudinal bundle. In the first group descending degeneration was seen in both posterior longitudinal bundles passing to the antero-lateral columns of the cord, in which it could be traced to the lumbar region. Much fine (terminal) degeneration was found round the anterior horn cells, especially in the cervical and lumbar enlargements. There was very little ascending degeneration in this group; any which did occur appeared to be associated with a tunnelling forwards of the lesion into the lower pons. In group  $\beta$  the descending degeneration was in all respects similar to that just described, but in addition there was here a very marked ascending degeneration affecting the more mesial and ventral parts of both the posterior longitudinal bundles. The third and fourth nuclei contained many fine degenerated fibres; no change, however, was observed in these nerve trunks.

In group  $\gamma$  there was marked descending degeneration in the right posterior longitudinal bundle, passing to the antero-lateral columns of the cord, where it was reinforced by the degenerated fibres of the direct spinal tract from Deiters' nucleus. There was a smaller amount of degeneration in the left bundle, which reached



the antero-lateral columns of the cord but disappeared below the cervical enlargement. Ascending degeneration was very marked in the left posterior longitudinal bundle, while a few altered fibres appeared in the right bundle also. This degeneration could easily be traced to the third and fourth nuclei, but in no case did it reach these nerve roots.

In group  $\delta$  the lesion destroyed the sixth nucleus, cutting across the genu of the facial nerve in doing so, so that both sixth and seventh nerves were degenerated. Many degenerated fibres could be traced across the raphe into the posterior longitudinal bundle or sixth nucleus of the left side. The fibres passing from Deiters' nucleus to the posterior longitudinal bundle (Ramon y Cajal) were of course cut by this lesion. The descending degeneration here was very similar to that in group  $\gamma$ , the right bundle was chiefly affected, but a few degenerated fibres were seen in the left one also; through the bundles the degeneration passed into the spinal cord, in which it could be traced (on the right side) into the lumbar region. Fine degeneration was visible amongst the anterior horn cells, especially in the cervical and lumbar regions. Ascending degeneration was found in both posterior longitudinal bundles; but while it occupied the mesial and ventral part of the left bundle as in group  $\gamma$ , it lay in the more dorsal and lateral portion of the right bundle, where it probably consists of fibres connecting the sixth to the fourth and third nuclei. Fine degeneration was present in the third and fourth nuclei, but, as before, the nerve trunks were intact. On comparing the results obtained in these various groups, it is obvious that the posterior longitudinal bundle cannot be regarded as being formed entirely of either ascending (Kölliker, Tschermak) or descending (Gehuchten, Held) fibres; the nature of the degeneration depends entirely on the level of the lesion and the amount of damage inflicted on the fibres derived from Deiters' nucleus. These fibres from Deiters' nucleus take a large part in the formation of the bundles, descending in the homolateral strand to the spinal cord and ascending in the heterolateral bundle to the mid-brain. The relation of the descending fibres to the anterior horn cells in the cord is clearly shown in all these cases, as also that of the ascending strands to the ocular nuclei. The direct passage of fibres from the posterior longitudinal bundle into the third and fourth nerve trunks, first stated to occur by Duval and Laborde and recently reaffirmed by Gee and Tooth, was not found to occur in any of the cases examined.

The relations of Deiters' nucleus are of interest in respect to the large part its fibres take in the formation of these bundles:—Blumenau and Tschermak have pointed out its important connections to the sensory (posterior) columns of the spinal cord;

Bruce and Turner have shown its relations to the cerebellum; while Sabin and Thomas have defined its connections with the vestibular nerve. The importance of the relationship between Deiters' nucleus and the posterior longitudinal bundle would seem to suggest that through this bundle are distributed influences arising in Deiters' nucleus as the result of stimuli reaching this cell mass from the cerebellum, the semicircular canals or the sensory strands of the spinal cord. So that to the functions already ascribed to the posterior longitudinal bundle may be added that of co-ordinating eye and body muscle nuclei for the purpose of adjusting the balance of the body in response to cerebellar, vestibular or sensory stimuli. AUTHOR'S ABSTRACT.

**ON THE LOCALISATION OF A RESPIRATORY AND A CARDIO-  
(255) MOTOR CENTRE ON THE CORTEX OF THE FRONTAL  
LOBE.** J. W. LANGELAAN and D. H. BEYERMAN, *Brain*, 1903,  
p. 81.

It has been pointed out by Winkler that concentration of attention is in presumably normal persons accompanied by definite respiratory and cardiomotor phenomena. Expiration and to a less extent inspiration becomes shorter and more superficial, and the thorax finally takes up an inspiratory position. On the relaxation of attention a compensatory retardation of respiration sets in, again chiefly affecting expiration with a tendency to deeper expiration at longer intervals. The cardiomotor changes consist of acceleration of the pulse during attention, and retardation afterwards. Another phenomenon of concentrated attention is the occurrence of small extensory movements of the fingers and neck.

Attempts were made by the authors to produce these phenomena concurrently or separately by stimulating a definite part of the cerebral cortex in dogs. They found that a weak faradic current applied to the top of the sigmoid gyrus where the coronal and presylvian fissures meet, accelerated respiration and led to the thorax taking up an inspiratory position. In some dogs the movements could be obtained separately; stimulation of the anterior and upper part of the area mentioned produced acceleration of respiration, while stimulation further back brought about the inspiratory position of the thorax. The area of cortex which produced these results borders on the region to which Ferrier ascribes stretching of the neck and movement of the head to the opposite side. By slicing away the cortex and stimulating the cut surface, the paths followed by the two impulses were found to diverge: that leading to the inspiratory position of the thorax was followed into the internal

capsule and pyramidal tract; while the fibres which on stimulation accelerated respiration were traced in front of the head of the caudate nucleus into the anterior limb of the internal capsule. Schüller found that they went as far as the median nucleus of the optic thalamus.

Another set of experiments consisted in the extirpation of this cortical area on one side. Immediately after operation respiration became irregular, greatly retarded, and interrupted by deep sighs. Sleep appeared to suspend these changes and they gradually passed off.

The experiments to localise cardiomotor changes were less successful, but the authors argue from work done by Winkler and Richet that it is at least probable that there is a cortical cardiomotor centre situated at the top of the sigmoid gyrus in the dog's brain.

The clinical history of four patients suffering from hysteria and epileptic fits is recorded, and curves showing changes in the cardiac and respiratory rhythms are appended. In one patient who had a tumour in the left frontal lobe, part of the second frontal convolution bordering on the anterior central sulcus was removed by operation, and one of the most noticeable features after the operation was the irregular respiration occasionally interrupted by sighs. The respiratory curve in this case closely resembled that obtained from a dog in which a similar operation had been performed. Similar phenomena were found in a boy operated on for epilepsy caused by an old injury over the median frontal convolution.

In the cases of hysteria, respiratory and cardiomotor changes were found during the attacks which bore a strong resemblance to the changes artificially brought about in dogs; suppression of the fits was attained in one case by educating the patient to breathe slowly and deeply when the attack was coming on.

The authors believe that in man a cardiomotor and a respiration-accelerating centre is situated at the base of the median frontal gyrus near the place where this borders on the anterior central gyrus.

PERCY T. HERRING.

**THE DESCENDING TRACTUS TECTO-SPINALIS, THE NUCLEUS  
(256) INTRA-TRIGEMINALIS AND LOCALISATION IN SPACE.**

O. KOHNSTAMM, *Neurolog. Centralbl.*, Nov. 11, 1903, S. 514.

THE secondary motor or co-ordination tracts are constantly claiming a greater clinical and physiological importance. The least understood of these is the tractus tecto-spinalis, which has hitherto been generally accepted as consisting of axons of cells in the

anterior quadrigeminal bodies which cross in Meynert's decussation and run in the opposite predorsal longitudinal bundle to the ventral columns of the cord. K. has been unable to find in this region cells in reactionary tigrolysis after hemisection of the cervical cord, but saw such in the mesencephalic trigeminus nucleus, so assumes that this is the only nucleus in the roof of the mid-brain which sends fibres to the cord. He finds corroboration of this view in the observation of van Gehuchten that in fishes fibres pass caudalwards from this nucleus in the dorsal longitudinal bundles; and in the experiments of Pavolow and of Münzer and Wiener, who could not find degenerated fibres entering the cord after destruction of one anterior quadrigeminal body, but only when the mesencephalic V nucleus was also destroyed. The tractus tecto-pontalis also arises in this region.

K. adopts the view of Helmholtz and Exner that localisation in space is the result of the association of the specific visual perception with a kinæsthetic perception from the ocular muscles, and the unconscious memories of the latter which may be known as determinants, are constantly utilised in such localisation. The kinæsthetic determinants of the movements of the eyeballs commence in the sensory trigeminus nucleus which receives sensory impressions from the oculomotor muscles, and from here is conducted to the cerebral cortex.

Then from some point of the central nervous system which is connected with the retina a tract must pass to some point on the course of the kinæsthetic determinant. This may be either in the forebrain by the association tracts between the occipital and central gyri, or in the midbrain, as K. thinks more probable, as in it the individual points of the retina are more discreetly represented. The anatomical connection of the primary optic centres of the midbrain with the sensory trigeminus nucleus is by the tractus tecto-bulbaris, and through it each time a definite spot in the retina is stimulated the corresponding kinæsthetic determinant is awakened in the sensory V nucleus, and from here is passed up to the forebrain cortex, where associated with the specific visual perception it makes possible the localisation of the visual perception in space.

The primary optic centres are also connected by the tractus tecto-pontalis with Deiter's nucleus, a centre for the co-ordinated innervation of the conjugate movements of the eyes.

Similarly tactile localisation is the result of combination in the cortex of the specific tactile perception and its kinæsthetic determinant (muscle sense) which ascends in the dorsal columns of the cord.

The majority of the fibres of Meynert's decussation are those of the tractus tecto-bulbaris which do not reach the cord.

GORDON HOLMES.

## PATHOLOGY

**THE NERVE ROOT AND GANGLIA LESIONS OF TABES.**

(257) ANDRÉ THOMAS et GEORGES HAUSER, *Nouv. Icon. de la Salpêtrière*, Nos. 4 and 5, 1902.

THE work of these authors on the pathogeny of tabes is on much the same lines as that of Nageotte in his various papers on the subject since 1894. Their conclusions are, however, somewhat different from his. According to them there are three possible modes of origin of the sclerosis of the posterior columns: (1) it may be due to a primary degeneration of the nerve fibres; (2) it may be a secondary result of an affection of the posterior root ganglion; or (3) it may be secondary to damage to the posterior roots themselves. Practically speaking, no changes have been found in the ganglion itself, but a meningitis has been frequently found, and some observers have considered this a most important cause, either by strangling the roots at their entrance into the cord (Obersteiner and Redlich), or by compressing them a little before their entrance into the posterior root ganglion (Nageotte). Another theory has been more recently advanced by Massary, who, in view of the degeneration of both ends of the neurone found often in tabes, considers that it is due to a systematised disease of the centripetal protoneurone. Similarly Leyden and Goldscheider compare the tabetic process to that of an ascending neuritis from a peripheral nerve to the roots and spinal cord. (Still more recently Marie and Guillain have brought forward evidence to show that the initial lesion in tabes is a syphilitic lesion of the posterior lymphatic system of the cord.)

Thomas and Hauser have studied the posterior roots in a number of cases of tabes, both within and on either side of the root ganglion, and they find that one of Nageotte's points in evidence of an interstitial inflammation of the third portion of the posterior root, close above the ganglion, the *nerf radicaire*, namely, increase in the number of the nerve bundles, may be a physiological variation and due to splitting of the root higher up. In all their cases there was pachymeningitis, more or less marked, around the posterior root, and continued on to the perineurium of the root. Hyperplasia of fibrous bundles was seen, with thickening and hyaline degeneration of the coats of the small vessels, giving rise to small hæmorrhages in places. The arachnoid is similarly affected, and becomes fused to the dura mater. In addition there is seen an intense perineuritis of both anterior and posterior roots, just above the ganglion, between it and the serous cul-de-sac, the *nerf radicaire*, at the spot where Nageotte demonstrated an inflammatory lesion. The degenerative process

of the posterior roots is a simple atrophy, the myelin sheath and axis cylinder disappearing gradually and slowly, that of the former being the more rapid, while numerous thin axis cylinders remain. There is marked degeneration of the *nerf radicaire* which extends within the proximal third of the ganglion, but the peripheral pole of the ganglion and the peripheral nerve beyond it are quite normal. In many fibres the degeneration seems to be segmental, in places the fibre being large and again becoming small, as may be seen in the toxic form of neuritis due to lead, and in experimental lesions. They consider that the usual form of atrophy of the posterior roots in tabes is a segmental atrophy, a form which may be seen just as well in primary lesions of the cell or fibre as following injuries or inflammatory lesions of nerve fibres. Moreover, as many authors have shown, peripheral cutaneous nerve lesions are common in tabes, and they are also of a segmental nature. The theories of tabes which have had in view only the degeneration of the posterior roots appear to be too exclusive in neglecting the cutaneous nerve changes. It is remarkable that the degeneration appears in the same form in the two branches of the same neurone, the principal difference being that the degeneration of the posterior root runs more rapidly to the centre. They explain the absence of changes in the ganglion cells secondary to the lesion of the posterior roots as on all fours with Lugaro's and van Gehuchten's experiments, in which section of the posterior root is followed by no *réaction à distance* in the ganglion cell, while section of the peripheral end does produce it. The result of their researches is that they refuse to accept Nageotte's lesion, viz., interstitial neuritis of the *nerf radicaire*, as of capital importance in the production of the tabetic degeneration. They conclude that the fundamental lesion in tabes is a neuritis comparable to that of toxic inflammations, such as lead neuritis, but showing a predilection for attacking the posterior roots, and on account of its slow progress and slight tendency to recovery it appears more like a primary degeneration than inflammatory in character. To explain this special predominance of the degeneration upon the posterior root fibres, one may call in other factors, such as Nageotte's transverse neuritis, which would cause vascular alterations in the posterior root system. Possibly a functional weakness of the ganglion cell may help in producing greater degeneration of its central than of its peripheral fibre; or possibly a general infection of the sub-arachnoid cavity may make the cerebro-spinal fluid in tabetics toxic, and thus affect the posterior roots.

(Nageotte in a footnote at the end of his paper in *La Presse Médicale*, 3rd Jan. 1903, in replying to a criticism of Thomas and Hauser that his theory did not explain the peripheral nerve

changes, remarks that in the sympathetic lesions it is the large fibres which are attacked, and that they are derived from the cord, and therefore pass through the *nerf radicaire*. The peripheral nerve changes he divides into two groups: those of the motor nerves, due directly to the neuritis affecting the anterior as well as the posterior roots; while the neuritis of the cutaneous nerves he conceives to be cachectic in nature, and predisposed to by the fact that one end of the neurone fibre proceeding from the ganglion cell, viz., the *nerf radicaire*, is already damaged by the neuritis, and that thereby the vitality of the peripheral fibre is diminished. He suggests also that numerous forms of neuritis such as lead or alcoholic may occur quite independently in tabes.)

WILFRED HARRS.

**THE CYTO-DIAGNOSIS OF TABES.** WIDAL, SICARD and RAVANT, (258) *Rev. Neurol.*, No. 6, mars 30, 1903, p. 289.

At the last meeting of the Neurological Society of Paris, MM. Armand-Delille and Camus reported that out of thirteen cases of tabes, under the care of M. Dejerine, only four showed a distinct lymphocytosis in the cerebro-spinal fluid. The authors of this present paper, together with M. R. Monod, announced in January 1901 that they had constantly found a distinct lymphocytosis in the cerebro-spinal fluid of cases of tabes, general paralysis, and other chronic diseases of the nervous system. These results were corroborated by MM. Babinski and Nageotte, who found the test positive in twenty-five out of twenty-six cases of tabes, and in four cases showing nothing abnormal, except the Argyll-Robertson sign. Other observers have confirmed their statements also in cases of general paralysis. In support of their contention, the present authors have examined other thirty-seven cases of tabes, in only one of which was there any doubt about the existence of a well-marked lymphocytosis. They therefore re-assert that lymphocytosis is practically constant in tabes. In criticising the results obtained by MM. Armand-Delille and Camus, they observe that the different results can only be explained by a difference in technique, and insist that, in order to make comparisons with their observations, their methods of procedure must be scrupulously followed in every detail. They recapitulate their method of performing the test. Lumbar puncture is performed in the usual way, and from 3-6 c.c. of cerebro-spinal fluid are collected into a tapered sterilised tube. It is this tube which should be centrifugalised, as pouring the fluid from one glass to another should be carefully avoided. Having centrifugalised for ten minutes, the tube is inverted, and the fluid allowed to drain completely away. The tube must be kept inverted, so that no

fluid can run back from the sides of the tube to dilute the deposit which remains of course in the tapered point. A very fine capillary pipette is then to be passed into the tip of the tube and moved about, when the last dregs of the deposit will be sucked up into the pipette by capillary attraction alone. Only by this means can the most concentrated preparation of the deposit be obtained. There may be no visible deposit to the naked eye, but any cells present will pass into the pipette together with the small quantity of fluid which remains in the tip of the tube even after inversion for some time. The contents of the pipette are then gently expelled and distributed over two or three slides in small drops, which should not be spread out over a larger area than 2-3 mm. sq. The films are then dried at room temperature, or at 37° C., fixed in alcohol and ether, and stained by any ordinary method—*e.g.* thionine, hæmatein and eosine, etc.

In normal cerebro-spinal fluid so prepared, lymphocytes may be entirely absent. At most, one or two will be found in each field of the microscope with an oil immersion lens. For a distinct positive reaction at least six to ten should be found in many fields. The authors indicate that they do not regard this lymphocytosis as indicating more than a simple advancing irritation as opposed to an acute congestive or inflammatory condition, which shows itself rather by the presence of an excess of polymorpho-nuclear elements. So it is found in such conditions as tabes, general paralysis, tubercular meningitis, where one meets a reaction on the part of the meninges. One sees this lymphocytosis also in cases of syphilitic meningitis and in syphilitic meningo-myelitis. According to the authors an even slight lymphocytosis in a hemiplegia ought to strongly suggest a syphilitic origin. In ordinary hemiplegia from hæmorrhage, or softening immediately after the onset of the condition, one may find a certain number of polymorpho-nuclear cells, but these are gradually replaced by round mononucleated elements, and this condition does not persist, and is not marked. In syphilis of long standing, where the disease is quiescent, the cerebro-spinal fluid is usually normal. A lympho-cytosis (in a syphilitic patient) indicates a progressive lesion, and should always put the clinician on his guard, even though there may be no other apparent sign or symptom of the disease. The authors have obtained some evidence that certain manifestations of syphilis, apart from the nervous system, may be associated with a spinal lymphocytosis. They have noted it in three cases of tertiary ulceration of the palate. In the secondary stage of the disease, even in the absence of cephalalgia or other nervous symptom, there may be a lymphocytic reaction of the meninges, thus indicating the susceptibility of the nervous system to the syphilitic virus. Cerebral tumours coming to the surface



and irritating the meninges may apparently cause a lymphocytic reaction. No such reaction apparently results from a central neoplasm. In uncomplicated cases of Pott's disease, neurasthenia, "classic polyneuritis," lymphocytosis is absent. It has been demonstrated, however, in cases of zona, and in certain cases of sciatica. Its absence has been noted in many infective diseases, infantile and adult, *e.g.* typhoid, erysipelas, smallpox, even where spinal pain was marked; a curious exception has been noted in its presence in cases of mumps (in three out of eight cases). It has also been noted in some cases of pneumonia with delirium.

In conclusion, the authors believe that the cytological examination of the cerebro-spinal fluid is of the greatest importance to clinicians, revealing as it does a meningeal lesion in many chronic advancing nervous maladies, and often aiding materially in differential diagnosis.

STUART M'DONALD.

**OBSERVATION ON ASCENDING DEGENERATING FIBRES IN  
(259) THE PYRAMIDAL TRACTS, WITH REMARKS ON MARCHI  
PREPARATIONS.** KARL PETREN, *Neurolog. Centralbl.*, 1903,  
S. 450.

IN a case of incomplete transverse lesion in the upper dorsal portion of the cord, the result of a trauma, from which the patient died 2½ months later, degenerated fibres were traced upwards in the site of the direct and crossed pyramidal tracts as far as the pons varolii, where the examination ceased.

Similar degeneration above the lesion has been described by Stewart and by Thiele and Horsley, but the latter were inclined to regard the degeneration as descending and the result of the cerebral concussion received at the time of the accident. Such cerebral concussion can be excluded in this case, so the degeneration must be regarded as ascending.

GORDON HOLMES.

### CLINICAL NEUROLOGY

**POLYMYOSITIS.** H. OPPENHEIM, *Berliner klin. Wchnschr.*, 1903,  
(260) No. 17 and 18.

SINCE this affection was first discussed in 1887 by Wagner, Hepp and Unverricht, many studies of it have been published, of which the chief are those of Strümpell, Loewenfeld, Senator, Fraenkel, Lewy, Lorenz, and Kader. It is therefore surprising that it is not now more generally recognised. The author knows of only one reference in French (by Lépine), and one in English, by Gowers, who says in the end of 1899, "I do not know whether this disease has yet found a place in English medical literature and systematic teaching."

The author has seen and treated more than twelve cases and upon these he bases his observations.

The disease usually develops in an acute or subacute manner. Common prodromal symptoms are: Disturbance of general health, sinking feelings, heaviness in the limbs, headache, shivering, gastric disorders. Then, as the first symptom of involvement of the muscles, pain, more or less intense, is complained of either in circumscribed areas such as one calf, or in several positions at one time, particularly in the limbs. This pain may be tearing, pulling, or boring in character, and is increased by active or passive movement. Active movement is affected to such a degree by the inflammatory and degenerative changes in the muscles, that in severe cases patients are almost entirely crippled, and lie helpless in bed unable to move a limb. In slighter cases the paresis remains confined to certain muscles. Deep pressure and palpation of the substance of the muscles causes unbearable pain.

When the disease is fully developed we may note the following features. The patients are generally confined to bed and complain of pain on every attempt at movement. There are fever, physical weakness, and generally hyperidrosis and affections of the mucous membranes.

The face is usually swollen, especially about the eyelids.

The amount of swelling of the limbs varies. It may be slight or may give the limbs an unformed appearance. It affects chiefly the proximal parts of the limbs, but this is not a definite rule. There may be swelling of the backs of the hands, of the thighs, or of the abdominal region. The character of the swelling varies also, the feeling presented on palpation being sometimes that of œdema, at others that of a firm compressed infiltration.

The swelling is generally found over the muscles which are most affected. It seems to disappear on palpation. It may be absent, or may disappear, or may be less extensive than the affection of the muscles.

In the muscles, alteration in consistency is the principal change. The muscle is often weaker, and may be softer than normal, so that apparent fluctuation may be present. More commonly, and especially in the later stages, the observer is astonished by the increase of consistency and the solidity of the muscle on palpation. This may be so great that the muscle presents the resistance of a board to the fingers. The muscles may become matted together, and even adhere to the bones, especially in cases of so-called interstitial myositis.

There is also an inclination to contracture in the affected muscles, and this may be noticed early. The author found it most often in the biceps.

Muscular atrophy usually appears first in the later stages.

Single muscles may be wasted, while in others the inflammatory swelling may be the most prominent element.

Electrical excitability is diminished or absent.

The tendon reflexes may be exaggerated in slight cases. More commonly they are diminished, and sometimes abolished.

The skin is usually affected, and this has led to the disease being denominated dermato-myositis. The eruptions vary in character, and have been compared to purpura, roseola, urticaria, herpes, erythema nodosum, and eczema. Scaling and desquamation may lead to dermatitis. In some cases the skin may be glossy.

The mucous membranes are also affected, the most common lesions being stomatitis and angina. In Struppler's case death was caused by œdema of the larynx.

The heart may be involved, and such symptoms as tachycardia, and arrhythmia have been noticed.

Of complications nephritis is the most common. The involvement of the mucous membranes is very common and very important. The author is so impressed by this feature that he has coined the name dermato-mucoso-myositis for this form.

The differential diagnosis of polymyositis from sclerodermia is often difficult. As a rule sclerodermia has an insidious onset and a chronic course, polymyositis is generally acute or subacute. Polymyositis may, however, last a year or even far longer.

The diagnosis from trichiniasis is also difficult. The prognosis is uncertain. It is usually considered very unfavourable, but in the author's experience of ten cases of generalised polymyositis and dermatomyositis only two ended fatally; in five there was complete or almost complete recovery.

As to treatment, the author recommends energetic diaphoresis, hot air baths, packing in woollen blankets, hot drinks and aspirin. The patient should be sweated in this way for at least an hour every second day. Alternately, or in the second period of treatment, thermo-massage is used, and later simple massage, gymnastics and electro-therapeutics. The author is convinced that such treatment leads to improvement or even recovery.

W. B. DRUMMOND.

**NOTE UPON A POSSIBLE RELATIONSHIP BETWEEN CARCINOMA (261) AND NERVE OR TROPHIC AREAS.** G. L. CHEATLE,  
*Brit. Med. Journ.*, 1903, p. 904.

In this paper Cheatle points out the remarkable relationship which in many cases exists between the distribution of the primary growth in cancer and certain well-recognised nerve areas. He is

also inclined to think that the incidence of cancer in a nerve area is not a fortuitous circumstance but may be due to nervous influence, direct or indirect. In this respect some cases of cancer are analogous to herpes, morphœa, moles and nævi, all of which frequently map out sensory root-areas, cranial or spinal. Certain infective processes are also particularly common in the "naso-labial" area around the mouth.

Cheatle's thesis is most strikingly illustrated by examples of rodent ulcer, many of which he figures. He shows that there is a remarkable tendency for rodent ulcers to localise themselves to the area supplied by the fifth cranial nerve with no lesion elsewhere. The skin supplied by both fifth nerves may be completely destroyed without the growth extending into the territory of the cervical nerves (Fig. 1). The fifth nerve area of one side, or the area of one or more adjacent divisions of the fifth nerve (Figs. 2 and 3), may be marked out by rodent ulcer. When the growth starts at the meeting-place of two contiguous divisions of the fifth nerve, these two areas tend to be mapped out before the disease spreads to others. A growth starting in the middle line tends to map out the corresponding divisions of both sides, producing a bilateral symmetrical lesion. Rodent ulcer often starts at the spot where a branch of the fifth becomes cutaneous, this being most typically exemplified in the infra-trochlear nerve. Other cases of rodent ulcer are figured, in which the area of the cervical spinal nerves is mapped out whilst the fifth nerve escapes (Fig. 4). It is rarely that a rodent ulcer starting in the fifth area spreads into the cervical area, or *vice versa*.

Hutchinson's crateriform ulcer, which may or may not be a squamous epithelioma, frequently arises on the points at which nerves become cutaneous. The same, according to Cheatle, may be said of typical squamous epithelioma. Cancer of the lip often maps out the "naso-labial" area, which is a compound area in which the infra-orbital, buccal and mental nerves participate. Cancer beginning on one side of the tongue tends to stop short at the middle line.

His second main point is that there is some reason for supposing that carcinoma may originate in a nerve area as a result of the nervous influence over that area. Whether such influence is of the nature of excessive or diminished nerve function, he does not state. The clinical fact remains that multiple rodent ulcers have a singular preference for the area supplied by the fifth cranial nerve. He hints that cancer may not be due to the mere influence of infection and emphasises the fact that cancer belongs essentially to the degenerative period of life. Bearing in mind that peripheral irritation can induce visible changes in the cells of the posterior root-ganglion, he excised one of the root ganglia for the relief of



FIG. 1.—Rodent ulcer in first and second divisions of both fifth nerves.



FIG. 2.—Rodent ulcer in first division of fifth nerve.



FIG. 3.—Rodent ulcer in auriculo-temporal area of third division of fifth. The disease commenced in front of the pinna.



FIG. 4.—Rodent ulcer in area of cervical spinal nerves; not implicating area of fifth nerve.



agonising pain in an inoperable cancer of the breast. On staining with thionin, certain of the nerve-cells in the excised ganglion were found to have the Nissl granules broken up and the nucleus situated eccentrically. Whether these changes were due to the cancer *per se*, or to the mere irritation, or whether they preceded the carcinoma, he is at present unable to say. At the same time he recalls the clinical experience as to irritation being a great precursor of cancer. Carcinoma often appears in tissues such as moles which are associated with disorders of growth, and these moles are themselves associated with definite nerve-areas.

In conclusion he claims to have shown "that there is reason to justify us in taking into consideration the possibility that the genesis and spread of cancer, even when considered apart, may be connected, directly or indirectly, with the nerve influences which preside over the areas affected."

PURVES STEWART.

**TRAUMATIC MUSCULO - SPIRAL PARALYSIS CURED BY  
(262) OPERATION RELEASING THE NERVE FROM THE  
VICATRICIAL TISSUE. CHARLES K. MILLS and J. WILLIAM  
WHITE, *Univ. of Penn. Med. Bulletin*, March 1903.**

THE musculo-spiral paralysis in this case followed a fracture of the humerus about its middle. Before operation there was complete paralysis of the muscles below the elbow supplied by the musculo-spiral nerve, with abolition of faradic excitability and reaction of degeneration. Sensation was unaffected excepting in an area one and a half inches in diameter over the back of the hand, the metacarpal articulation of the index and middle fingers, and over the back of the thumb. Four months after the accident the patient was operated on by Dr White. The nerve was found firmly bound down by dense adhesions and tightly attached to the callus, although not surrounded by it. The nerve was separated from the surrounding structures with difficulty. Four weeks after the operation the extensor muscles of the forearm had in large degree recovered, the area of anæsthesia was disappearing, the patient was steadily improving, and there was every indication of complete recovery.

EDWIN BRAMWELL.

**ON A CASE OF TUMOUR OF THE SPINAL CANAL (DORSAL  
(263) SEGMENT). F. RAYMOND, *Journ. de Neurol.*, May 1903.**

THE record of this case is prefaced by a few introductory remarks upon the subject of spinal tumours in general, the whole being written in lecture form.

The patient described was a young man who had had malaria at the age of 16; two years later he began to drag his left leg, and

within a few months the right leg was also affected, so that he had to take to his bed. It was at this stage that he was first seen by Raymond; the patient was then found to have paralysis of both legs, with contractures, exaggeration of tendon-jerks and double extensor plantar reflexes, with absence of the other skin reflexes in the affected parts; there was complete anæsthesia below the umbilicus, with a zone of hyperæsthesia above this.

The sensory signs improved a little during the next few months; tactile anæsthesia remained absolute on both sides, but sensation to pin-pricks, heat and cold, had become incomplete on the right leg, remaining complete on the left. Eventually the anæsthesia spread upwards, so as to reach the level of the xiphisternum. There was paralysis of defæcation and urination. There was no vertebral column deformity nor pain on percussion. The pupils did not dilate when the legs were pinched, but did react to stimuli from the upper extremities. The patient never had root pains at the level of the lesion.

The patient was operated upon, but died of hæmorrhage. The lesion was found to be at the sixth dorsal segment; it was a very vascular ramifying sarcoma invading the cord. The histological details are to be published later.

The discussion is necessarily discursive rather than full, and the reasons for operation are not given in detail. The introductory observations are valuable rather as a brief survey of the whole subject than for any new matter they contain.

STANLEY BARNES.

**TWO CASES OF SARCOMA OF THE SPINAL CORD.** Von (264) H. SENATOR, *Sonder-Abdruck aus den Charité-Annalen*, xxvii. Jahrg.

SENATOR records two cases of sarcoma of the spinal cord with an autopsy in each case.

CASE I. A female, aged 69, was admitted to Senator's Klinik on August 21st, 1901, complaining of complete inability to move the legs, with a feeling of tingling and numbness and painful contractions in them, a burning feeling in the feet, and retention of urine.

Ten months previously she first noticed tingling in the great toes and pains in the legs. The knees then began to feel stiff. The pains which became more frequent were especially severe in the hip joints. The feet used to swell. For six weeks prior to admission she had had the greatest difficulty in walking. The patient's previous health had been good and there was no history of syphilis.



*State.*—A well-nourished woman. No glandular enlargement and no fever.

The special senses, cranial nerves and upper extremities were in every respect normal. The patient was quite unable to make any movement with the lower extremities, which were rigidly extended, the feet being dorsiflexed. The rigidity was greater on the left side. The knee-jerks were very active, right > left. The Achillis-jerks and foot clonus were not obtained. The Babinski sign was marked on both sides, right > left. Stroking the sole of the foot produced clonic spasms in the muscles of the lower extremities and abdomen. The abdominal reflexes could not be obtained. There was relative tactile anæsthesia and inability to distinguish between slight differences in temperature, and pain over the lower extremities. Pin-pricks were well felt except over the left thigh. The upper border of the disturbance of sensibility reached as high as Poupart's ligament. At a later date there was complete anæsthesia and then analgesia over the legs, with a zone of tactile anæsthesia extending, on the anterior aspect of the body, from Poupart's ligament up as far as the upper border of the eighth rib. There was no spinal deformity, but L. 1, L. 2, and L. 3 were tender on pressure; on a subsequent occasion the lower thoracic vertebræ up as far as D. 6 were tender on pressure.

After three weeks in hospital L. 1 and 2 became very prominent and there was marked shortening of the trunk, so that the costal margin reached almost to the iliac crest. Flexor spasms became very troublesome and the lower limbs gradually took up a position of marked flexor contracture. The patient developed a bed sore in November and died on March 9th, 1901.

At the post-mortem a tumour of the dura mater about the size of a pigeon's egg was found about the level of D. 7 to D. 9.

The tumour appeared to the naked eye to be a sarcoma. No operation was attempted because of the age of the patient and because the tumour had been diagnosed as a carcinoma.

Senator remarks that the spinal deformity and shortening of the trunk were not dependent upon a destruction (*zusammensinken*) of the bodies of the vertebræ, but appeared to him to depend upon muscular contracture, in particular contracture of the abdominal and pelvic muscles, the recti abdominis, quadrati lumborum, ileo psoas and others.

CASE II. A female, aged 67, admitted on May 11th, 1902, complaining of sacral pain and loss of power in the legs. The first symptom had been pain over the sacrum and between the shoulders, which was often so severe that the patient was unable to move. For four weeks the feet and abdomen had been swollen. Fourteen days before admission the legs became suddenly weak, the weakness being especially marked in the left.

The patient was a well-developed woman, but rather anæmic.

There was great loss of power in the legs and slight rigidity. The knee-jerks were lively on both sides. There was no distinct foot clonus. Babinski's sign was well marked on both sides. Tactile anæsthesia was present over the trunk as high up as the nipple; over the anæsthetic area painful impressions were incorrectly localised. Marked hyperæsthesia existed on the outer side of both feet. There was great spinal rigidity but no deformity, but D. 2-4 were very sensitive to pressure. The patient complained of pain in the legs, and there was incontinence of urine and fæces and cystitis.

She died three days after admission, evidently as a result of general infection secondary to the cystitis. At the post-mortem there was an acute cystitis with necrotic areas on the mucous membrane of the bladder. An extradural tumour the size of a date was found on the left side of the cord at the level of D. 3 and 4. To the naked eye the tumour appeared to be a fibro-sarcoma.

In neither case was there a microscopical examination of the tumour or cord, the specimens being kept for purposes of naked-eye demonstration.

EDWIN BRAMWELL.

**SOME CONSIDERATIONS ON THE PATHOGENESIS OF**  
(265) **SYDENHAM'S CHOREA.** GIANASSO, *Rif. Med.*, Anno xix,  
No. 16.

THE author first refers to the numerous theories which have been brought forward to explain the causation of chorea, and after mentioning the theory of multiple cerebral embolisms, and the dyscrasic theory, which considers the chorea to be a consequence of anæmia and other diseases, he deals more in detail with the three theories which are now most supported, the rheumatic, the infective and the nervous.

The rheumatic theory suggests that chorea consists in a diseased condition of the nerve centres, which is directly dependent on the rheumatic infection, a cerebral localisation of rheumatism which may or may not be accompanied by articular or muscular symptoms. But a history of rheumatism is frequently absent in cases of chorea, and some observers look on the occurrence of rheumatism with chorea as a pure coincidence.

The infective theory is based on the frequency with which the infective diseases precede or accompany the chorea, on the complications which are met with in chorea major (arthropathies and endocarditis), and on the fever which accompanies the disease. Some authorities say it is due to a specific organism; others that it may be produced by various organisms or their toxins. Pianesi

described a bacillus which he isolated from a case of chorea major, and staphylococci and streptococci have also been found. Changes in the nervous centres similar to those found in the acute infections have been described.

But these facts, while they explain the cases of chorea major, do not solve the question of chorea minor, and the author suggests that cases of chorea major are due to infections complicating the latter disease, but not the cause of it.

With regard to the nervous theory, Sturges says that chorea is due to psychic excitation, Stevens to errors of refraction of the eyes; others say that it is due to excitation of the basal ganglia. These views are not generally accepted.

Charcot, Joffroy, and Leroux consider it a neurosis which causes psychic, motor, and sensory disturbances, which may disappear without leaving any persistent anatomical modifications. Joffroy says it is a neurosis of evolution; Oddo agrees with this, but insists on the importance of a degenerative taint.

The nervous theory, however, does not explain the articular pains, the neuralgias and the fever. Moreover, neuroses manifest themselves in the most various ways, quite unlike the cyclic course of chorea.

The author has examined forty-six cases of chorea minor, and has come to the conclusion that the idea of an infection acting on a predisposing ground, the degenerative taint, is the most probable explanation of this disease, and he considers that chorea major is caused by a secondary infection.

R. G. Rows.

**PSYCHIC DISTURBANCES OF CHOREA MINOR IN RELATION  
(266) TO THE MOTOR DISORDERS.** BUCCELLI, *Riv. Speriment. di  
Freniatria*, Aug. 1902.

THE cardinal symptom of chorea is a morbid functioning of the central motor apparatus; but with this, psychic changes, which are the direct consequences of the motor disturbance, are always associated, and in some cases other psychic disorders are met with which are only indirectly connected or perhaps simply modified by the disease.

This perversion of the psycho-motor function exercises an injurious effect on all mental processes.

The author dealing in this paper with these mental changes shows first the effect of the motor disorder on the will. The will can no longer be considered to be some superior and spiritual power, but the resultant of organic physical conditions. Phylogenetically and ontogenetically the will is the highest development of the

primitive faculty of the nervous system, the sensory-motor reflex, and this highest ideo-motor reflex is governed by the same laws as the lower automatic reflexes.

Moreover, we cannot conceive of will without the idea of co-ordinated movements, movements in which the lower, less complex activities are subordinated to the highest or ideo-motor function.

It would be expected, therefore, that when the motor factor of the will is disturbed as it is in the choreic, in whom the subordination of the lower activities to the higher is so much diminished, the volitional power would be interfered with.

This is confirmed by clinical observation, and it is found that the patient who exhibits the choreic movements, is indolent, indifferent, capricious. In some cases these mental symptoms actually precede the motor disturbance.

Next to this morbid condition of the will the most important change is loss of attention, both voluntary and involuntary. This is not a simple coincidence, but is a necessary result of the disorders of the motor sphere.

The motor element of attention, the exclusive intellectual state with spontaneous or artificial adaptation of the individual, is, by psychologists, considered to be an indispensable condition of attention rather than a phenomenon consecutive to it; and it must be present so that the relative monoideism, which is the essence of attention, may be maintained.

In the choreic the involuntary and often diffuse movements make the physiological process of attention impossible.

Connected with this as almost necessary consequences we find a certain amount of obtuseness and incompleteness of perception, a weakening of the power of discerning and analysing. The processes of association are disturbed, and the memory is weakened. Recent events are remembered incompletely, giving rise to the condition which has been called "lacunar memory."

Lastly, there is a disturbance of the emotional side of the patient, with rapid changes from one emotion to another which may have an entirely opposite character. In choreics, too, there is often a slightly melancholic condition which by some is thought to be connected with loss of volitional energy. This condition may, however, depend somewhat on the general intoxication of the organism, which is now regarded as the cause of the disease.

An important point mentioned is that choreics are frequently marked degenerates, and they therefore possess less resistive power to outside influences, and are more easily exhausted.

The type of psychic disturbance described above as a result of the motor disturbance is observed in most choreics. But mental changes are often seen in the disease, which can have only an indirect connection with the fundamental process. The degenera-



tive factor is a strong predisposing influence to their development, and there can be no doubt that they are also influenced by the continuous morbid movements. Among these a hypochondriasis and a slight mental confusion with hallucinations and illusions are the most frequent.

Acute hallucinatory delirium is sometimes added to the chorea, and is probably due to a secondary intoxication independent of that which caused the chorea.

In some cases, the author says, the chorea runs the usual course for a time, and then there is a sudden outburst of acute delirium, whilst in others the delirium and the choreiform movements develop simultaneously. He suggests that in the former series there is a secondary infection, and that in the second series we are not dealing with true Sydenham's chorea, but with cases in which the choreiform movements are produced by the same intoxication which causes the psychic disturbances. This is often seen in cases of pellagra (Dubini).

With regard to the association of chorea and hysteria, the author says that it is usually the case of hysteriform disorders occurring in choreics.

These disorders are common to all degenerates, and degeneracy is very frequent in both hysterical and choreic subjects.

Hysteria itself is now considered a psychosis and not a neurosis, and the various anæsthesias, the capriciousness, and the disorders of the memory, can best be explained in both affections by considering them to be the result of some disturbance of attention.

The individual value of these various pathogenic factors will vary with the predisposing constitution of the patient and with the choreic process. Experience of cases shows that when the hysterical disorders are dependent more on the constitution of the subject they appear at the beginning of the chorea, or even before the characteristic movements are seen. The outburst is generally acute, sudden, and the symptoms show themselves in various ways. When, however, they are due to the choreic process, they appear much later, more gradually, and are less typical.

In the first series the hysteria frequently persists after the chorea has disappeared, maintaining itself independently for some time; in the second series it disappears with the chorea. The symptoms also in the first series can be influenced by suggestion; in the second they are very little, if at all, affected by suggestion.

R. G. Rows.

**A CASE OF MYASTHENIA GRAVIS COMPLICATED BY ANGIO-  
(267) NEUROTIC OEDEMA. THEODORE DILLER, *Journ. Nerv.  
Ment. Dis.*, April, 1903.**

**AFTER** referring to the recent literature of this disease the author mentions Oppenheim's diagnostic criteria of Myasthenia, and then describes the following case which occurred in a married woman of 29, the mother of three children. The patient's family history and previous health had been good, but she was addicted to alcohol. Two years previously she had been confined to bed for five months on account of general debility and nervous prostration, and though she had improved she never completely recovered. On July 11, 1902, she had a chill followed by difficulty in articulation. She was admitted on July 14 with a temperature of 103.8 which gradually dropped but rose again on July 31st, on which day large circumscribed areas of oedema appeared on the right shoulder, outer side of right elbow and forearm, over the crest of the ilium and on the outer side of the right leg. These patches were painful but not inflammatory, and disappeared in five days. On August 7, 1902, patient's condition was one of general weakness, specially marked in the face, palate, and muscles of articulation. The speech was slurred and became eventually almost unintelligible after she had talked for some time. Her face had a mask-like appearance owing to the immobility of the muscles. There was no muscular wasting or secondary changes and the reflexes were sluggish. There was occasional diplopia, but the author could not produce exhaustion of the muscles of mastication though any exertion caused general weakness. The diagnosis was based on the general condition of the patient taken along with the special affection of the bulbar group of muscles and their easy exhaustion and the absence of any organic, motor or sensory symptoms. No electrical tests were applied. The association of angio-neurotic oedema with myasthenia has not as far as the author knows been previously recorded.

T. GRAINGER STEWART.

**AMNÉSIE ANTÉROGRADE CONTINUE, TOPOAGNOSIE ET  
(268) TROUBLES DE LA PSYCHORÉFLECTIVITÉ ÉMOTIVE,  
CONSÉCUTIFS À UN CHOC MORAL. E. DUPRÉ, *Rev.  
Neurolog.*, May 1903, p. 449.**

**THE** interesting case here recorded is that of a man of 53 with a three years' history of hæmoptysis and physical signs of phthisis. A sister is an idiot and a daughter has symptoms of mental

derangement. He had an attack of meningitis at the age of ten, and had for years been a heavy drinker and smoker. There was no evidence of syphilis.

Three and a half years before he came under observation he had a severe mental shock and since indulged to excess in absinthe. It was at this time that the particular mental symptoms described appeared. These consisted essentially in the absolute impossibility of fixing and retaining sensorial images and consequently of utilising them in his psychical processes by association with the well-preserved mnemonic images of the past. All sense impressions were perceived, understood and rationally valued, but nothing was retained, so that he never knew aught of what had happened in the previous few minutes. At the same time his memory of all that occurred previous to the onset of this condition was remarkably good. The deficit was purely amnesic and in no way intellectual.

The result of this condition—psychical blindness to all sense impressions—was a complete inability to orientate himself in time or in new surroundings or to learn to know and again recognise people he had met since its onset. Thus though he could give a thorough description of his native town as it was in his childhood, he remembered nothing of the changes which had been made in it previous to a recent visit.

He was conscious of this amnesia.

He was also somewhat morbidly emotional, but of much greater intensity was the emotional psycho-reflex affection. Any slight excitement—examination, conversation—produced somatic expressions of awe and fear without the corresponding psychical state, that is, there was a disproportion between or even disassociation of, the emotions and their expression.

Besides well-marked general arteriosclerosis there were few abnormal physical signs. The pupils were unequal and contracted sluggishly to light and there was a slight degree of optic neuritis. His gait was of the cerebellar type, like that of a drunken man; the deep reflexes were brisk but the plantars were of the normal type. He complained of headache, and an excess of lymphocytes were present in fluid obtained by lumbar puncture.

The diagnosis given is "hystero-organic encephalopathia," the organic basis of which may be a chronic meningitis with cerebral arteriosclerosis, and possibly disseminated vascular lesions in the cortex and subcortical regions consequent to the mental shock.

GORDON HOLMES.

**A CONTRIBUTION TO THE CORTICAL ORIGIN OF TREMORS.**(269) R. MASSALONGO, *Rev. Neurolog.*, May 1903, p. 455.

THE present paper is based on a case of lung tuberculosis which while in hospital developed rapidly fatal tubercular meningitis.

On the first day on which symptoms of the latter complication appeared, fibrillation and fascicular contractions resembling the myokimia of Schultze and Kny were frequently visible in the various muscles of the limbs and face. On the next day this was more pronounced, but the contractions were not yet violent enough to cause limb movement. All voluntary movements were still preserved but were slightly ataxic. Later the patient became delirious, and the muscular contractions were so severe as to cause involuntary limb movements of a choreic or athetoid character. Several general epileptiform attacks preceded death, which occurred on the fourth day.

At the autopsy tubercular meningitis was found localised to the frontal and parietal convolutions, and tubercle and small foci of softening in the subjacent cortex. Otherwise the nervous system was normal.

This case is published as a support to the view for long held by the author, that in the cortical motor cells is the exclusive origin of tremors, and that extra-cortical lesions can only produce such indirectly by reflex action of the cortex; that in other words, the cortex is the single "seismogenic centre." The histological examinations of other observers on cases of various natures are cited in support of this view, and as further argument is brought the frequent occurrence of myoclonia and other tremors in epilepsy, in fright and other psychical states, and in hysteria, undoubtedly conditions of cortical origin.

Irritations of the psychomotor cortex varying in intensity, duration and extension are capable of producing the various forms of myoclonia, and the possibility of their combinations and transformations depends on the sensibility and impressionability of the different nervous elements as well as on their nature and quality.

GORDON HOLMES.

**A CASE OF INTENTION-TREMOR, WITH REMARKS ON THE**(270) **PATHOGENY OF TREMOR.** Dr DE BUCK, *Journ. de Neurol.*, 20 mai 1903.

THE author describes the case of a man aged 36, whose symptom-complex consisted of vertigo, volitional tremor most marked in the right arm, weakness of the lower limbs, slight nystagmus and an equally slight impairment of speech—apparently a *fruste* form of disseminated sclerosis. The remarks on the pathogeny of volitional



tremor are unsatisfactory, inasmuch as they consist almost entirely of quotations; but the view the author takes is that intention-tremor is centripetal in origin, in nature approaching ataxia, and dependent on a lesion in the medullo-cerebello-cortical paths; whereas tremor during repose is centrifugal, is expressive of an alteration in muscular tone, depends on lesions in the extra-pyramidal tract, and may be classified with hyperkinetic movements such as chorea and myoclonus. S. A. K. WILSON.

**THE TENDO - ACHILLIS - JERK AND ITS CLINICAL SIGNIFICANCE.** ARTHUR VON SARBÓ. Karger, Berlin, 1903

THE author has examined the tendo-Achillis-jerk in (a) 300 patients none of whom were suffering from any nervous affection, and (b) 884 cases of nervous disease. The following is an abstract of his conclusions:—

1. The tendo-Achillis-jerk, like the knee-jerk, can always be elicited in healthy individuals.
2. Exaggeration of the tendo - Achillis-jerk is met with especially in functional neuroses, in alcoholism, general paralysis, and in nervous states associated with anæmia and malnutrition, and affords evidence in association with other symptoms of an exaggerated reflex excitability in these diseases. In a number of healthy persons, however, the tendo-Achillis-jerk is lively; exaggeration is of as little importance in differential diagnosis as is exaggeration of the knee-jerks.
3. In some cases of cerebral hæmorrhage, an exaggeration of the tendo-Achillis-jerk on the paralysed side is apparent before any change in the activity of the knee-jerk is demonstrable. It is probable that a similar condition will be observed in the case of other forms of brain disease, *e.g.* tumour, if attention is directed to the point.
4. Loss of the tendo-Achillis-jerk has the same diagnostic significance as loss of the knee-jerk:—(a) unilateral loss points *ceteris paribus* to a neuritis (sciatica, alcoholic neuritis, syphilitic, etc.) or poliomyelitis (or unilateral or disseminated process in the spinal cord or roots) at the level at which the reflex passes through the spinal cord; (b) bilateral loss of the reflex points *ceteris paribus* to tabes or general paralysis or to polyneuritis.
5. Cases of tabes and general paralysis are met with in which the tendo-Achillis-jerk disappears before the knee-jerks; if general attention is directed to the examination of the tendo-Achillis-jerk the early diagnosis of tabes and general paralysis will be distinctly advanced.
6. In cases of neuritis of the lower limbs the condition of the

tendo-Achillis-jerk is of value in forming an opinion as to the probable duration of the condition.

7. Where the examination of the knee-jerk is difficult (stout people, etc.), the tendo-Achillis-jerk may afford valuable information.

8. In every neurological examination the tendo-Achillis-jerk is to be examined as well as the knee-jerk. EDWIN BRAMWELL

**CONTRIBUTION TO THE STUDY OF THE ACHILLES-JERK (272) AND THE FRONT-TAP.** G. L. WALTON and W. E. PAUL, *Journ. Nerv. and Ment. Dis.*, June 1903, p. 341.

THE authors in a short paper record a series of observations based on an examination of 500 persons either apparently healthy or suffering from some non-nervous affection. They have also examined thirty-five cases of tabes and some other cases of nervous disease. The following are the conclusions arrived at:—

- "1. The Achilles-jerk is practically as constant in health as the knee-jerk. This reflex varies less in health than the knee-jerk in excursion and activity, and is the most easily elicited and uniform of all tendon reflexes."
- "2. The Achilles-jerk disappears, as a rule, early in tabes dorsalis, and its absence is as diagnostic of that disease as is loss of the knee-jerk. We have not seen a case far enough advanced to establish tabes with persistence of the Achilles-jerk, except one case in which both the knee-jerk and the Achilles-jerk were present on one side only. We have observed bilateral preservation of knee-jerk and loss of Achilles-jerk in two out of five cases of tabes."
- "3. Enfeeblement of knee-jerk in health on one side or both may be due to prior toxic influence, as diphtheria. This may also be true of the Achilles-jerk, though in the one case in which it could be demonstrated of the knee-jerk, the Achilles-jerk was normal. Further observations on this point are desirable."
- "4. The front-tap is present (generally on both sides) in about 40 per cent. of individuals in ordinary health; in some it is very active. It follows that its presence alone, even if active, does not establish disease, nor indicate excessive irritability of the nervous system."
- "5. In organic disease the front-tap is generally increased with the other reflexes in hypertonic, and decreased (generally wanting) in hypotonic states."
- "6. In the so-called functional disorders, hysteria, neurasthenia, and unclassified psychoses, we have found the front-tap present in 71 per cent. of cases. In epilepsy we have

found it present in 75 per cent. of cases. The test may therefore here prove of aid in combination with other findings, though its mere presence or even activity is not of positive diagnostic value, nor does its absence negative the existence of neuropathic conditions."

- "7. Both these reflexes deserve to be placed upon the list of routine tests for purposes of diagnosis. This is particularly true of the Achilles reflex, which is of the greater positive diagnostic value."

EDWIN BRAMWELL

**VASCULAR AND TOXIC ACROPARÆSTHESIA, OR SENSORY  
(273) DISTURBANCES OF THE EXTREMITIES OCCURRING  
INDEPENDENTLY OF PRIMARY DISORDER OF THE  
NERVOUS SYSTEM. H. BATTY SHAW, *Practitioner*, June  
1903, p. 756.**

THE writer of this article gives a brief description of several cases which came under observation, and in which the patient complained more particularly of various disorders of sensation affecting the extremities, more especially pains, formication and numbness. In some cases such symptoms were the only ones complained of, in others various trophic disturbances were present as well, such as wasting of the connective tissues, transverse ridging of the nails, and thickening of the skin, the latter being so marked that sensation was blunted. In most of the cases no direct evidence was forthcoming of any nerve lesion, nor was there any wasting of muscles; cramps, however, were met with, and spasm of the muscles of varying degrees of intensity were occasionally seen. In most of the cases the symptoms were such as to lead the observer to conclude that they were not primarily due to neuritis, but that they depended either upon an organic vascular lesion affecting arteries and possibly veins, or that they were due to toxic agents acting on normal vessels, and then producing symmetrical disturbances, or upon vessels which were diseased in one extremity or part more than in the corresponding region on the other side of the body, with the result that the vascular spasm was sufficient only in the former localities to produce some nutritional and functional disturbance in the nerves distributed to these regions. These results may be temporary and pass off when the toxic agent is removed; in others the lesion in the nerve is permanent, and in support of this latter view, microphotographs are given, showing interrupted degeneration of small nerve fibres taken from a fatal case of tetany in an infant, the preparations having been made by Dr Sidney Martin, Professor of Pathology at University College, London.

The writer further points out that though such grouping of sensory disturbances is very commonly met with, and is in most cases a distressing symptom, it yields to treatment more or less readily. According as there is vascular disease or not, the symptoms may usher in many grave disorders; and he points out that one of the cases described terminated a few days later in death from uræmia, due to granular disease of the kidneys. Another case gradually developed the well-known symptoms of gastric tetany, and also proved fatal; and a third case was found to have such extreme gastrectasis and gastropstosis that the stomach was found lying in the suprapubic area. This latter case was treated by posterior gastroenterostomy, and the symptoms described were cured.

This group of symptoms has been described as "acroparæsthesia" by Schultze, and many writers have met with them in a variety of disorders of extremely varied nature, and the purpose of this contribution is to draw attention to the importance of the symptoms and to their pathogenic relationships. Many of these cases improve, so that there is no opportunity of examining the nerves and vessels, or central nervous system; but the writer has endeavoured, by calling attention to the very similar signs and symptoms met with in Raynaud's Disease, in Erythromelalgia and Sclerodactyly, to prove that exactly similar symptoms are met with without any changes of the central or peripheral nervous system, and that in most cases vascular disease can be demonstrated; and where this fails, that functional vascular disease can be invoked to explain the nervous disorder. Toxic agents have been investigated in connection with gastric tetany by Bouveret and Devic, by Halliburton and by Carnegie Dickson, and it is argued that where organic vascular disease is absent, toxic agents can produce, at first, various paræsthesiæ dependent upon purely disturbed nerve function, such as are met with in mild cases, or even paræsthesiæ dependent upon structural organic nerve lesions, such as have been discovered by Dr Sidney Martin in infantile tetany. The contribution closes with an account of various methods of treatment for the alleviation of this distressing group of symptoms, and with a list of references to the bibliography of the subject.

AUTHOR'S ABSTRACT.

**THE EFFECT OF MENTHOL ON THE CUTANEOUS NERVES.** Dr (274) JOTEYKO, Brussels, *Journ. de Neurol.*, 20 mai 1903.

It is well known that the application of menthol to the skin, as for instance in the shape of antimigraine pencils, produces in a few seconds a sensation of intense cold. It was employed by Goldscheider to differentiate the properties of sensory nerve

terminations in the skin. He found that the temperature of the part to which the menthol was applied, so far from being lowered, was actually raised a little, and came to the conclusion that this substance had the remarkable quality of stimulating the ends of those sensory nerves which convey sensations of cold. The mere touching of the "mentholised" area, even by objects not cold, such as one's finger, produced this feeling of intense cold also.

The author undertook to examine the action of menthol on all the four cutaneous sensations, cold, heat, pain and touch, attempting more especially to explain the admitted analgesic action of this substance. He experimented on the skin of fifteen students, employing for the purpose Chéron's Algesimeter, an instrument which registers in tenths of a millimetre the depth in the skin of a metallic point sufficient to produce a minimal sensation of pain. Analyses are given of various experimental results which lead the author to maintain that the first effect of menthol on the skin is to diminish the sensibility to pain; it then excites the nerve terminations conveying sensations of cold, and when the analgesia reaches its maximum the sensation of cold is also at a maximum. The analgesia then disappears, while the sensation of cold is followed by a general depression of the nerves conveying ordinary tactile sensations: and finally, only if the application of the menthol has been very complete, is there tingling or even a feeling of warmth, indicating that the nerve terminals associated with the conduction of heat sensations are being stimulated. It would appear then that menthol depresses the touch and pain nerve terminations, while it stimulates those for heat and cold. This may perhaps be explained by the anatomical consideration that according to Von Frey the nerves of cold and of heat are more deeply situated in the skin than the others.

S. A. K. WILSON.

**THE SKIAGRAPH IN GROSS BRAIN LESIONS.** HENRY K. (275) PANCOAST, *University of Penna. Med. Bul.*, March 1903, p. 37.

THIS paper records the results obtained by the Skiagraph in a series of eight cases of cerebral disease. Four of the cases were diagnosed as cerebellar tumour, two were cases of hemiplegia, one was a Jacksonian epilepsy, and the last idiopathic epilepsy. In only two of the cases were the results verified by operation, and in not one by post-mortem examination.

Generally, the results were not very satisfactory; and the author concludes that the value of the Roentgen rays in lesions of the brain is at present doubtful. He thinks, however, that with increased experience in normal as well as in abnormal cases that it might become in time quite a useful method of diagnosis.

W. K. HUNTER.

## PSYCHIATRY

**PSEUDO - HALLUCINATIONS (PSYCHIC HALLUCINATIONS OF (276) BAILLARGER).** A Contribution to the Psychology of "Demenza Paranoide." E. LUGARO, *Riv. di Patol. Nerv. e Ment.*, f. 1, 1903, p. 1.

THE observations of the author are based on a very complete examination of eight cases illustrating the points to be commented on, and at the beginning of the paper there is a resumé of the literature on the subject.

Specific characters of pseudo-hallucinations. From his cases the author concludes that in chronic delirium there are presented certain states of consciousness which consist in mental images deprived entirely of sensorial characters, either of hearing, sight or muscular sense. They are thus purely psychic hallucinations and consist usually in representations of heard words (internal voices transmitted by mysterious means), but can also be simple images, more or less dissociated from the corresponding verbal images; in ideas, in thoughts, or in representations of acts (direct suggestions, not verbal).

A common character is the incoherence or the antagonism which these representations bear to the remainder of the consciousness, and this is the reason why they are held as something extraneous to the personality of the patient, as extraneous voices, as thoughts and as the will of others. They are always recognised as a fact morbid and quite exceptional. Even when associated with true hallucinations the patient can always detect the pseudo-hallucinations as subjective phenomena, quite devoid of the objectivity which is the characteristic of true hallucinations. Owing to this subjective characteristic Lugaro prefers the term pseudo- to psychic-hallucination, as the sensations are always appreciated by the patient as subjective facts transmitted by mysterious artifice from one personality to another.

Pseudo-hallucinations and hallucinations of hearing. True hallucinations of hearing are characterised by objectivity. The patient hears the voice coming from outside just as if someone talked to him as in ordinary conversation. In pseudo-hallucinations the patient may speak of the voices he hears but they do not give him the impression of being heard by the ears. They are quite subjective and are often described by him as mental voices suggesting certain acts.

Pseudo-hallucinations and epigastric voices. In the greatest number of cases the phenomenon of the epigastric voice depends on pseudo-hallucinations of heard words accompanied by hallucinatory sensations in the abdominal and thoracic viscera, in which

the voices are localised by the patient. In one of Lugaro's cases the epigastric voices were accompanied by sensations in the parts to which the voices were referred—in the viscera and in the fingers. The voice was aphonic, *i.e.* mental. The patient talked incessantly, but his words were merely responses to the voices or independent of them altogether. He imitated with his voice and gestures the person who appeared to be speaking within, but these words and gestures were always part of his own personality and not that of the person who spoke in his mind and interfered with his viscera. Even the phonetic apparatus can be the seat of hallucinatory sensations, but such a localisation is not necessary for the manifestation of the epigastric voice.

Pseudo-hallucinations and psychomotor hallucinations. As hallucinations can occur in any sensory area the muscular sense of the phonetic apparatus can be disturbed, and thus from a hallucination in this region there arise psychomotor verbal hallucinations. The hallucination may be very little in evidence and is only inferred from the movements of the tongue and lips, and from the abnormal sensations in these parts, which demonstrate nothing else than the incipient formation of verbal motor impulses. But these impulses are not always psychomotor hallucinatory. They can be associated with hallucinations of every kind and can even be present as a normal phenomenon which is not hallucinatory. Again in certain patients the voices may not be in the mouth but even in other parts of the body. These hallucinations may not be pure sometimes, the patient giving indications of hearing voices and even distinguishing them by their tone.

But a pure psychomotor verbal hallucination only gives to the patient the illusion of speaking without hearing the sound of his own voice, because the excitation in the psychomotor verbal centre is projected to the organs of phonation and not elsewhere; every other localisation is an index that the hallucination is not psychomotor verbal.

The essential condition therefore in psychomotor verbal hallucinations is a hallucination of the muscular sense of the phonetic apparatus, and one can exclude that they form the basis of the phenomenon of pseudo-hallucinations. It is probable, according to Lugaro, that many of the cases described as psychomotor verbal hallucinations may be only cases of pseudo-hallucinations of heard words associated with hallucinatory sensations in the phonetic apparatus or with verbal impulses, which hallucinations and impulses determine the reference of the pseudo-hallucinations to this apparatus with a mechanism analogous to that by which the location of the epigastric voice is brought about.

Pseudo-hallucinations and the substitution of an extraneous personality for that of the patient (*sdoppiamento*). Pseudo-hallucinations usually cause a delirium of possession and therefore a *sdoppiamento* of the personality. This is different from the *sdoppiamento* of personality which one finds in somnambulism, spontaneous or induced, in which the two personalities are entirely or in part unconscious of each other. In the case of pseudo-hallucinations the conviction of the *sdoppiamento*, of a trebling, or of any multiplication whatsoever of the personality, is only an effect of the delirient interpretations.

The strange, the enforced thoughts, the speeches formed as by another person and the interruption or disturbance of the normal course of spontaneous association make the patient believe that other personalities speak in them. If the pseudo-hallucinations are associated with visceral hallucinations, then the extraneous personality is localised in the viscera. A slighter form exists where the external personality does not invade the patient but influences him from afar by mysterious means; even slighter where the means employed to disturb the patient's cerebration are purely physical or chemical (objective).

Pseudo-hallucinations, psychomotor hallucinations, and *il pensiero ad alta voce* (*Gedankenlautwerden*), or the thought expressed aloud. Lugaro rejects the theory that *il pensiero ad alta voce* can depend on pure psychomotor verbal hallucinations. These can only give the illusion of an aphonic repetition of thought on the part of the patient but not on the part of others. In true repetition of the thought there occurs a true hallucination of hearing, as there is clinical evidence to show that the hearing centre is affected, and that the true hallucination of hearing follows and repeats thoughts formed internally, *e.g.* a patient may hear whispered into his ear words or phrases that have passed through his mind. It is—as one of Lugaro's patients says—as if a little devil whispered and repeated to him what he thought. The phenomenon of echo or repetition of thought has certain relationships with pseudo-hallucinations. Some patients think that all their mind is known to others, that they can think of nothing without others knowing about it. Believing themselves in communication with other people by suggestion and other exceptional agency, by means of which the extraneous ideas are transfused into them, they hold that by this same agency their thoughts are drawn from them. Or, again, believing that their thoughts are of external origin, it seems natural to them that they must be known to the person who has the power of transmitting them. There is thus a delirium of transmission of thought which is a consequence of the subjective impression of the pseudo-hallucinations. This latter condition is, however, quite distinct from the pheno-



menon of the repetition of the thought accompanied by sound, for in the true condition there is a pure hallucination of hearing.

Pseudo-hallucinations and enforced ideas (*idee coatté*). The enforced character of pseudo-hallucinations and their separation from the personality, the fact that the patients themselves recognise them as strange or morbid, may induce one to think that there is a certain affinity between them and the so-called enforced ideas. The two conditions are somewhat different, however. Many authors affirm that these enforced ideas represent anything whatsoever extraneous to the personality to the patient, *i.e.*, phenomenon of psychic dissociation. According to Lugaro enforced ideas have always a more or less evident connection with the other ideas and are always evoked by the normal associative paths; their morbid character consists in the fact that are maintained in the focus of introspective attention by the affective states which accompany them and are their cause of origin, and they are strengthened more and more by repetition, *e.g.* the fundamental cause may be a morbid fear, and this affective state draws the forced idea into the field of attention and keeps it there. The idea does not arise unnoticed, but is expected and feared by the patient. It is not set free from the associative paths, but is mixed up in a true vicious circle of association. Pseudo-hallucinations are quite different, they are not provoked by any affective state and reach the patient's consciousness unnoticed. They may be of varying nature, and the patient is usually indifferent to them, and when they determine an affective reaction it is dependent more or less on the delirient ideas which are kept up by the pseudo-hallucinations, than on the pseudo-hallucinations themselves.

Pseudo-hallucinations, impulses, and enforced actions. Enforced acts are usually associated with enforced ideas and impulses, which are associated very frequently with pseudo-hallucinations. These impulses are either an inevitable consequence of mental verbal suggestions—to which the will of the patient cannot offer any material resistance—or arise spontaneously as deliberations extraneous to the personality of the patient, which only lends itself mechanically to the execution of them. They are unforeseen and surprise the patient, and usually consist in frivolous insignificant acts. Impelled acts dependent on impelled ideas are recognised, expected and feared, because they are always disagreeable, and sometimes awaken horror in the patient's mind (ideas of suicide or homicide). They may be curbed by the volition for a long time, but the struggle between them and volition is constant. The phenomenon of enforced speech (*parola coatta*, *Zwangsreden*) can be associated with and be part of pseudo-hallucinations of

heard words without psychomotor verbal hallucinations coming into play.

As regards the frequency of pseudo-hallucinations, Lugaro is of opinion that in chronic delirium they—especially of heard words—are more commonly met with than true hallucinations of sight or hearing. He is inclined to oppose the view that they are the precursors of true hallucinations, and thinks that more frequently patients may have true hallucinations at first, but later, when the mental symptoms are quieter, the hallucinations become purely representative—*i.e.* psychic.

Pathogenesis. Lugaro admits that the pathological anatomy underlying pseudo-hallucinations is very indefinite, but mentions what has been found in various forms of mental disease presenting them. In “*demenza paranoide*” lesions of nerve cells, increase of neuroglia have been observed. In G.P. and in senile insanity pseudo-hallucinations are sometimes seen, and in these diseases many irritative changes are found. Now in G.P., in which one often finds many of the symptoms of dementia præcox (catatonia, motor impulses, muticism, negativism, etc.), there is always present a very distinct lesion of the polymorphic layer, and this lesion is always more distinct in this region than in any other layer of the cortex; and in climateric melancholia, in which one can observe pseudo-hallucinations and frequently negativism, alterations of the cells and neuroglia proliferation have been seen in the deep layers of the cortex. The author advises, however, a study of the fine changes in dementia præcox before any definite conclusion is come to.

The following are some of Lugaro's conclusions not as yet mentioned:—Pseudo-hallucinations, impelled acts, and partly hallucinations, as well as the products of their combination (*il pensiero ad alta voce*, impelled speech with suggestion, epigastric voices), depend probably upon the same causal factor—*i.e.* on internal cerebral irritations which act apart from the ordinary mechanism of association, and determine therefore the presence of sensorial impressions, of word images, of volitional resolutions independent of the personality of the patient.

The specific distinction between pseudo-hallucinations and true hallucinations renders the hypothesis legitimate that these two phenomena are developed in different ways—*viz.*, the true hallucinations in the sensorial areas, and the pseudo-hallucinations in the associative centres.

While the theory of a local stimulus in the associative centres is sufficient to explain the origin of pseudo-hallucinations, the origin of complex hallucinations cannot be explained simply by the presence of a local stimulus in the sensorial centres. To explain these one must admit the additional action of the associa-

tive centres. Pseudo-hallucinations are characteristic of chronic psychopathic states. They are more frequent in "demenza paranoide," but can be observed in every form of dementia præcox. They are also seen, but with less frequency, in senile, climacteric insanity, and in G.P.

The mental disturbance characteristic of dementia præcox consists in a perturbation of the elaboration of the motives of action, of the volition, and of the conduct. On this depend the affective insensibility, the impulses, catatonia, negativism, catalepsy, etc. The pseudo-hallucinations are an immediate associative effect of such disturbance.

It is probable that the fundamental disturbance depends upon an elective and systematised lesion of special neurons. The system affected cannot be sensory or motor, because motion and sensation are unaffected, and as memory and ideation are preserved the system for association of images must be intact. The lesion must affect a system of neurons concerned with the highest co-ordination between representations, the corresponding emotions and the execution of the acts. From the data to hand at present obtained from cases presenting the psychic phenomena examined in his work, Lugaro is inclined to suspect that the system injured is contained in the deep polymorphic layer of the cortex. DAVID ORR.

**A CASE OF PERNICIOUS ANÆMIA WITH INSANITY.** By (277) Dr H. MARCUS, *Neur. Centralb.*, May 16, 1903, p. 453.

DR MARCUS here gives an account of an interesting case of pernicious anæmia associated with mental and other nervous symptoms. The patient, a man of 37, with a fairly good family history, but always slightly neurotic and peculiar, put himself on a course of treatment on account of excessive corpulence. After losing 14 kg. in weight he began to feel very weak. He continued his work, however, though at that time it was specially heavy. A month or two later he began to act in an insane way, quite contrary to his usual doings. He spent money recklessly, incurred heavy debts which he could not meet, and became very irritable and unreasonable. He was certified and sent to an asylum, and on arrival there it was noticed that he was very pale. On examining the blood the hæmoglobin was found to be only 35 per cent., and the red corpuscles to number 3·4 millions. Nervous symptoms were also present in the shape of muscular tremors, impairment of speech, and marked inco-ordination of movement both in the arms and legs. He was very restless, had exalted delusions and lacked common sense. For a month he was treated with iron, but no improvement took place. Indeed, the anæmia became distinctly worse. He was then put on arsenic, and a

decided and rapid change for the better immediately set in. In a few months more he was again quite well.

At the outset the symptoms very closely resembled those of general paralysis, but the subsequent course of the case appears distinctly to exclude this possibility. It is more likely that there was some affection of the nervous system secondary to the anæmia, as has already been recognised and described, and such as one occasionally sees after severe influenza, or else there was a fortuitous combination of physical disease with hereditary nervous instability. There has also to be taken into account the possible influence of the means used to reduce the patient's weight, though what this was is not stated.

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#### THE HALLUCINATORY DELIRIUM OF ACUTE ALCOHOLISM.

(278) CHARLES S. WALKER, *Am. Journ. Insan.*, April 1903, p. 583.

IN this communication a graphic account is given in the patient's own words of what he remembered of his experiences during the attack. The patient was a man of thirty-three years of age, suffering from acute alcoholism of four or five days' duration. Some of the more outstanding features of his description are:—Elaborate detail is remembered. Hallucinations referable to the auditory sphere preponderate, the hearing of singing being a specially marked feature. Visual hallucinations also occur, including visions of animals. The delirium has a systematising character, and there is strong suicidal tendency. The delusions and hallucinations were frequently of a painful or terrorising nature, but at times apparently pleasant, some of the singing being "lovely."

The patient was treated with rest in bed, no alcohol, occasional hypodermics of hyoscin. hydrobrom. in  $\frac{1}{16}$  gr. doses. He made an uneventful recovery.

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# Review of Neurology and Psychiatry

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## Original Articles

### UPON THE RELATIVE AFFECTION OF MUSCLES IN PROGRESSIVE BULBAR PARALYSIS.

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THE object of this paper is to point out the early affection of the supra-hyoid and infra-hyoid muscles in many cases of progressive bulbar paralysis, and to suggest that the dysphagia and dysphonia arising in the course of the disease are referable to the atrophic paralysis of these muscles rather than to affection of the intrinsic muscles of the palate, pharynx and larynx.

In the clinical examination of a series of cases of progressive bulbar paralysis and of amyotrophic lateral sclerosis, certain features have been noticeable in connection with the acts of deglutition and phonation, which cannot easily be explained on the ground of atrophic paralysis confined to the tongue, palate, pharynx and larynx.

When several cases presenting a similar degree of lingual atrophy, and a like condition of the palate and pharynx as regards reflex action, were compared, it was found that the severity of the dysphagia and dysphonia varied remarkably, being in some cases slight, in others severe, and in searching for some other factor to explain this discrepancy it was found that the degree of dysphagia and dysphonia which was present in each case varied directly with atrophy of the supra- and infra-hyoid muscles.

The reflex contraction of the pharynx and of the palate upon excitation was in many cases obtained more easily than in the healthy subject. In other cases it was produced less readily or with difficulty, but in all cases reflex contraction both of the palate and pharynx was obtained, and when obtained the contraction was complete, forcible and sustained.

Opportunity was afforded for comparing the more severe degrees of dysphagia among the cases of bulbar paralysis with dysphagia resulting from bilateral implication of the vago-accessory roots, in a case of neoplasm of the basi-occipital bone. The nature of the dysphagia seemed entirely different in the two cases. In the case of vago-accessory palsy the difficulty was obviously pharyngeal, and was least marked when liquids were swallowed; while nasal regurgitation invariably occurred. In the progressive bulbar cases the difficulty was always greatest with liquids. In swallowing semi-solids the chief difficulty lay in the forcing backwards of the bolus between the faucial pillars, and further it was obvious, on observing the lateral aspect of the neck during the attempt at deglutition, that the forward and upward displacement of the larynx which allows the protection of the glottis by the epiglottis, did not take place. Nasal regurgitation was never observed, nor was a history of its occurrence obtained in any of the cases.

Laryngoscopic examination showed no obvious defect in the movements and position of the vocal cords, except in one severe case in which marked bilateral abductor paresis was present. Aphonia was never present, but the phonation was confined to the production of the lower note of the middle register; the higher notes and the very low notes could not be produced. Upon comparing the movements of the larynx as a whole in the cases and in the healthy subject, it was found that in the cases of bulbar paralysis the movement of the larynx upward for the production of the higher notes, and downward for the production of the lower notes, did not take place. Moreover, there was an absence of the normal fixation of the larynx during phonation.

A careful examination of supra- and infra-hyoid muscles showed that reduction of phonation proceeded *pari passu* with atrophic palsy of the supra- and infra-hyoid muscles.

The results of the clinical observations suggested that atrophic paralyses of the palate and pharynx are rare rather than common

events, and that affection of the supra- and infra-hyoid muscles was apparently the cause of the difficulties in deglutition and phonation. Paralysis of the larynx is stated by many authorities to be rare in bulbar paralysis.

An examination of the various muscles concerned was made in several cases with the following results:—

#### HISTOLOGICAL EXAMINATION OF MUSCLES.

Ten of the cases ended fatally during my period of office as Pathologist at the National Hospital, and I am indebted to Dr Farquhar Buzzard for his kind permission to make use of another case recently examined by him. Of eleven cases, three presented the clinical aspect of progressive bulbar palsy without spasticity. The remaining eight cases were of the amyotrophic lateral sclerosis type. So far as the changes found in the muscles were concerned, no histological difference corresponding with the two clinical types was determined. The nature of the muscular change was the same in all the cases.

*The Tongue.*—The intrinsic fibre of the tongue, the superior and inferior lingualis, and the perpendicularis and transversus linguæ were the most severely affected of all the muscles. The palato-glossus, hyoglossus, genio-hyoglossus, and genio-hyoid were severely affected.

*Muscles of the floor of the mouth.*—The mylohyoid and the anterior belly of the digastric were markedly affected in all the cases. The posterior belly of the digastric and the stylo-hyoid were affected to a less degree.

*The Hyoid Muscles.*—The sterno-thyroid and thyro-hyoid were far advanced in atrophy, their condition corresponding very much with that of the intrinsic muscle of the tongue. The sterno-hyoid and anterior belly of the omo-hyoid showed extensive changes but of a less degree than the above.

*Muscles supplied by the fifth nerve.*—In the three most severe cases the characteristic changes were found affecting the angles of the muscle prisms; the atrophy was in an early stage. The mylohyoid and anterior belly of the digastric were severely affected.

*The Palate*—With the exception of the fibres of the palato-

glossus, the muscle of the palate presented no marked degree of atrophic change. The palato-pharyngeus was healthy.

*The Pharynx.*—In the majority of the cases atrophic muscular fibres were conspicuously absent from the pharyngeal wall. In the most severe cases a few patches of atrophic fibres were seen, but they bore a very small proportion to the number of healthy fibres. The fibres of the stylo-pharyngeus and of the palato-pharyngeus were unaffected.

*Laryngeal Muscles.*—In most of the cases there was either no affection of these muscles or a few atrophic ones could be seen at the angle of the muscle prisms. In the severe cases a more widely spread change was present, about one-fortieth of the fibres being atrophied. This atrophy was most marked in the crico-thyroid muscle.

The atrophic process was first noticeable at the corners of the muscle prisms, where several fibres were seen reduced to about one-fourth their natural size. The muscle substance of these fibres stained much more deeply than that of the healthy fibres, and the transverse striation was either absent or faintly marked. There was no sign of fragmentation or fatty change visible in sections prepared from formalin hardened material. The atrophic fibres appeared much more closely packed together than did the normal fibres, probably as a result of the greater shrinking of the former during the process of hardening. The muscle nuclei were greatly increased in number, both relatively and absolutely, in the atrophic fibres. The muscle spindles were conspicuous and the intrafusal fibres were not affected. There was marked increase of the endomysium and arterio-sclerosis in the atrophic area.

This atrophy of the fibres commencing at a corner of a muscle prism seemed to spread from fibre to fibre as if by contiguity, and in an even march over the muscle prism. Healthy and atrophic fibres were not scattered over the muscle bundles. The change did not commence on all the muscle bundles at the same time, for a prism in which all the fibres were atrophied was frequently seen side by side with a prism of healthy fibres.

The affection of the accessory muscles of deglutition and phonation in bulbar paralysis is revealed on clinical examination as follows:—The mouth cannot be opened actively or passively to the normal extent. On the attempt at laryngoscopic examina-

tion this becomes strikingly apparent. The masseter and temporal muscles show a considerable degree of contracture, presumably owing to the paresis of the opponents—the anterior belly of the digastric, the mylohyoid, the genio-hyoid, and genio-hyoglossus, all of which come into play in the act of opening the jaw against resistance. Unfortunately the external pterygoid muscles, which are of such great importance in the act of opening the mouth, were not examined in these cases; but the suggestion is allowable that they may be early affected.

If the observer, standing in front of the patient, place his thumbs, one upon either side, upon the submaxillary region, grasping the malar region with his fingers, ask the patient to open his mouth forcibly against this resistance, it will be noticed that the floor of the mouth does not harden against the observer's thumb—the digastric and mylohyoid are acting feebly. Similarly when asked to swallow, there is again no forcible hardening of the floor of the mouth—the mylohyoid is affected.

The tongue is found to be further back, and at a lower level than in a healthy subject. The hyoid bone and the larynx are also at a lower level than in health. I would suggest that the weight of the larynx, etc., and the absence of the support for those structures usually afforded by the supra-hyoid muscles, which are parietic in these cases, explains this position of the hyoid bone and larynx.

Such a position of the larynx must put the palato-pharyngeal muscles upon the stretch, and as these muscles tend to escape in the atrophy, a possible explanation arises, both for the low hanging palate commonly seen in bulbar paralysis, and for the characteristic nasal quality of the voice in cases where the presence of a brisk palate reflex proves that there is no marked atrophic palsy of that structure. That the palate does not act voluntarily in a normal way may be due in some cases to supra-nuclear affection of the pyramidal path supplying the palate, and in support of this argument the writer, using the Marchi method in several of these cases, has found many degenerate fibres in Pick's bundle (the pyramidal bundle supplying the nucleus ambiguus).

Atrophy of the infra-hyoid muscles is as a rule strikingly apparent when the anterior aspect of the neck is observed. There is a deep recess above the sternum between the lower ends

of the sterno-mastoid muscles. The inner edges of the sterno-mastoids are conspicuous, and the larynx is much less covered than in the normal subject. Often the isthmus of the thyroid body and all the supra-sternal rings of the trachea may be felt. The faradic excitability of the infra-hyoid muscles may easily be tested and their affection proved by the increased strength of current required and the feeble nature of the response. It is likely that these signs would be difficult to determine in subjects with well developed adipose tissue, but such a condition must be rare in bulbar palsy, for all the writer's cases were markedly wanting in adipose tissue.

The weakness of the supra- and infra-hyoid muscles is well demonstrated if the excursions of the larynx be watched during deglutition, and during the attempt to phonate a high and a low note, and compared with those of a healthy subject. If the hyoid bone be grasped between the finger and thumb of the observer during the attempt to swallow and phonate, its movements may be prevented with such pressure as gives the patient no discomfort, but in the healthy subject the hyoid bone cannot be so detained by the observer even though pressure such as to produce severe discomfort be used.

In a normal person, with a moderately thin neck, the prominence of the posterior belly of the omo-hyoid on the attempt to bend the head forward against resistance is well seen, but when there is paralysis of the infra-hyoid muscles this prominence does not occur.

The investigation of the nuclear affection and the nuclear representation of the affected muscles has involved a lengthy research at the present time incomplete. This will form the subject of a separate paper. It may be suggested here, however, that in a line with the nuclear affection in progressive muscular atrophy, bulbar paralysis is primarily and chiefly an affection of the hypoglossal nucleus and of its morphological continuation backwards into the first and second cervical segments—the ventral horn cells supplying the supra- and infra-hyoid muscles.

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**GRAPHIC APHASIA IN ASSOCIATION WITH EPILEPSY  
IN A CASE OF CEREBRAL TUMOUR LASTING  
NINE YEARS.**

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EPILEPTIC attacks often constitute one of the earliest symptoms of a tumour growing in or near the cerebral cortex, and these attacks are frequently of the Jacksonian type, spreading comparatively slowly and involving muscle-groups in regular sequence. In rare cases in which the tumour is situated behind the Rolandic cortex of the left hemisphere, attacks may occur in which aphasia is the prominent or perhaps the only sign. These attacks correspond exactly to the more usual form of Jacksonian seizures, and should the cortical discharge spread widely, a regular sequence of sensory and motor phenomena may come on. Dr Hughlings Jackson has often insisted that the careful study of the "march" of a fit is of the greatest importance, in that in this way it may be possible to trace out the physiological connections of the cortex. It is highly probable that an epileptic discharge does not spread anatomically but physiologically; in other words the discharge spreads not to those parts of the brain which are nearest to it in point of distance, but to those parts which are the most closely linked up in function and association with the discharging focus. The case recorded here was carefully observed over a considerable period of time, and the clear account given by the intelligent patient and his friends of other attacks adds further value to the record.

When first seen on March 14, 1902, Mr Alfred B. complained of inability to write for several months. He was a well-educated man, 37 years of age, holding a responsible post as cashier. His father had died at the age of 65, never having recovered consciousness after a stroke which occurred several months before his death.

*Previous history.*—At the age of 21 the patient had an attack of gonorrhoea, and at 28 he contracted a chancre which was diagnosed and treated as syphilitic; no secondary signs appeared.

He married at 30 and had had three children; the first two were alive and well, but the third was still-born.

*Present illness.*—About nine years ago (a few months after the chancre had disappeared), when apparently in good health, he went out to spend the evening with some friends. Whilst he was talking to them his speech suddenly became "queer"; the words he was saying were in English and were well-pronounced, but were strung together in such a way as to be incomprehensible; he was very garrulous, and his friends said afterwards that he seemed unable to understand them when they spoke to him. He did not try to read or write; he remained in this state for two or three hours and then suddenly fell down unconscious in a fit; he bit his tongue, but did not micturate or hurt himself. The fit (convulsions) only lasted a few seconds, and he then went to sleep, to wake up quite well the following morning.

A year later a second attack came on. He was out cycling at the time and a peculiar feeling suddenly came over him; he cannot further describe this sensation. He at once got off his machine and went to a stable near by; he tried to speak but could not; he signed successfully to a bystander to bring him a glass of water and said "Thank you" in reply. He could only partially understand what was said to him. When a doctor poured him out some brandy, he refused it (by signs) because he remembered being advised not to take alcohol in an attack. After this he gradually got worse and may have had a convulsion, but he is not certain. He recovered gradually, being able to understand speech before he could himself speak. The whole attack lasted about two hours and left him very "dizzy."

The third attack occurred two years later. The seizure came on suddenly; he was very dazed and quite unable to speak, but could partially understand what was said to him; he had no convulsion. He went to bed at once, and next morning on getting up had quite recovered. Since then, during the last five years, he has had several attacks, but all of them slight and of short duration. A year before he was first seen they averaged one a month, and after that gradually increased in frequency.

The recent attacks differ considerably from those described above; the following description was given by his wife:—After a few moments of uneasiness he goes "dizzy," but does not fall; he is at once unable to express himself accurately in words, but

can say some words, which he afterwards says he knows at the time are not the words he wants to use; for instance he has asked for "pen and ink" when he really wanted a "glass of water." At this stage his wife usually leads him to a couch. His eyes and head tend to roll to the right, and he becomes somewhat stiff in all four limbs. Numbness comes on in the right hand and extends to the right arm and side, and often to the right leg. If he attempts to walk he staggers a great deal, especially to the right, "as though he could not judge his distance." He often complains of a glaring light before his eyes, so that he can only see clearly on the left side. There is never any convulsion or loss of power, but if he is holding anything in his right hand he often drops it. He usually complains of headache after the attacks and then goes to sleep. In the slighter attacks, which occur now about every third day, the numbness of the right hand and arm are the most prominent signs.

Apart from the attacks his speech has gradually become awkward; he often hesitates for words, and sometimes uses wrong words. Letter-writing has been getting bad since Sept. 1901; this difficulty has been very pronounced, and now his letters are usually quite incomprehensible. He can read aloud well, except that he has difficulty with words at the right hand end of the lines. He appears to understand what he reads to himself.

He has had one or two "bilious attacks" lately, accompanied by headache; he has never complained of double-vision. Memory for recent events is decidedly impaired, but for distant events is good. His wife thinks he has become rather irritable lately.

*Status praesens* (March 1902).—He is a well-built man of 37, general aspect healthy. The thoracic and abdominal viscera show no abnormal physical signs. His disposition appears natural, and he was not irritable during a long clinical examination. He is not morbidly introspective.

*Aphasia, etc.*—As he speaks most of his words are perfectly articulated and rightly used, but occasionally he will stop—especially if he wants a rather uncommon word—and it may be several seconds before he thinks of it. Speech is a little slow, he says slower than it used to be.

He can understand perfectly every word that is said to him.

He can pick out rightly at once any of a series of objects before him when one is mentioned by name.

Most of the commoner objects around he can refer to by name; those less familiar to him he cannot name or does so with great hesitation; *e.g.* he could not name a "stethoscope," although he knew the instrument and its name; but when I mentioned in rapid succession the names of a series of objects, he at once stopped me when I said "stethoscope," recognising that as the correct name for the instrument before him.

He cannot read (to himself) as quickly as he used to do. This appears to be solely because he finds difficulty in making out the words on the right-hand side of the lines. He understands well what he reads.

He writes slowly from dictation, and very inaccurately. Few words of more than three letters are spelt correctly. The letters too are badly formed.

He copies from print to longhand very slowly. He often misspells words, frequently leaves out small syllables, and occasionally small words. He puts in no stops. He realises that the writing is bad, and often crosses out mis-spelt words.

In spontaneous writing he begins fairly well, but quickly tires out. The composition is bad, many words are crossed out, others are so badly written as to be illegible, and few stops are put in. He cannot write more than eight or ten lines in half an hour.

*Cranial nerves, etc.*—There is no cranial tenderness or abnormality. Smell is good both sides. Central vision is good in each eye ( $v=6/6$  both). There is no optic neuritis or atrophy.

*Fields of vision.*—The temporal field for the *right eye* is markedly contracted for all colours. The blind part of the field is crescent-shaped, the contraction being greatest in the horizontal axis, where the range is reduced to about half the normal. The nasal field is normal. For the left eye the nasal field is contracted about ten degrees, the temporal field being normal.

The other cranial nerves were normal.

In the limbs and trunk, power and the various forms of sensation were good. There was no astereognosis. The gait and reflexes were normal.

*Progress.*—No material change occurred until March 19th,

when one of his slighter attacks occurred. He was sitting out on a balcony talking to a friend when dizziness in the head and a sinking sensation in the epigastrium came on. There were no special-sense premonitory signs. He at once got up to walk to a couch and his friend noticed that he staggered and dropped his pipe from his right hand. By this time a sense of numbness in the right hand and leg had come on. When I saw him, about two minutes after the commencement of the attack, his speech was a little more hesitating than usual, but he used no wrong words and understood what was said to him. There was no objective sensory change (pain and touch). He now had a "shimmering in front of both eyes, like you see on a clear hot day at the seaside." This seemed to be as marked in the left visual field as in the right, and he "could see it in his mind's eye when his eyes were shut." His mouth felt dry on both sides, but was not really so on either. On the right cheek he had a surface sensation of prickly heat, but I could not detect any difference objectively between the two cheeks. There was no astereognosis. The right-hand grip was 95 and the left 105, a difference which could only be detected by the aid of the dynamometer. The pupils, reflexes and tendon-jerks were unaltered. The visual fields were then tested, *i.e.* about half an hour after the commencement of the attack, and when all his subjective sensations had passed off. It was found in comparing this chart with the previous ones that the field of the right eye had undergone a marked *concentric* contraction, and that there was a strong tendency to the spiral fatigue-curve of hysteria. There was slight general contraction of the field of the left eye also. By the following morning the fields had returned to their previous size. Slight headache followed the attack.

After this he gradually improved, and by May 11th could read much better and did not hesitate at all in speaking. His writing, however, was still bad (spontaneous, from dictation and copying). There was now no headache, vomiting or optic neuritis.

On May 21st "a bilious attack" came on. It began with frontal headache, and on May 22nd he vomited frequently and was almost unable to speak. On May 23rd the field of vision of the right eye was more contracted, but he was so dull that it could not be charted. On May 26th there was commencing

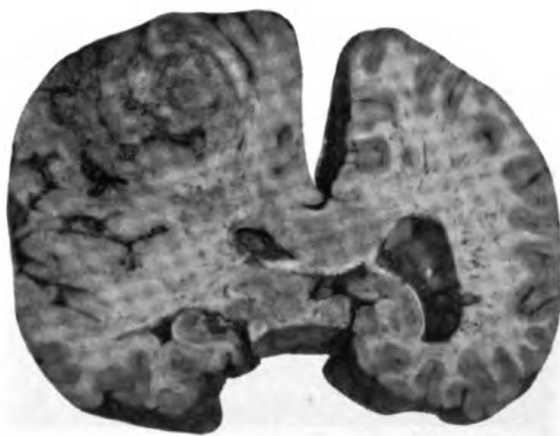
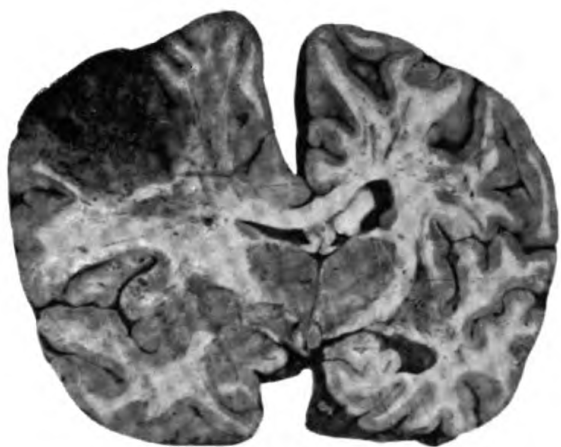
optic neuritis, with 3 dioptries of swelling in the left disc, and 2 dioptries in the right disc. He gradually became more and more dull, only rousing to complain of headache. Difficulty of respiration and cyanosis came on, and on May 29th he died of respiratory failure.

*Treatment.*—The treatment adopted was anti-syphilitic. Large doses of potassium iodide and of mercury were given for several weeks. For the headache phenacetin, antipyrin and antifebrin were used, the last-named being found most useful.

*Post-mortem examination.*—The brain alone was examined. All the convolutions were much flattened, and there was very little cerebro-spinal fluid present. The left hemisphere was much larger than the right, and presenting on the surface of the former in the parietal region was a growth whose superficial area was circular, and about the size of a crown-piece. The growth was bounded in front by the ascending parietal convolution, below by the supra-marginal, behind by the angular (which was partly involved by pink, young-looking growth), and above the superior parietal lobule was somewhat involved. The greatest diameter of the growth was at a depth of about 2 cm. below the surface; the basal ganglia were not involved. The growth appeared to be about the size of an orange. The anterior part of the tumour was soft, and contained extravasated blood; the brain immediately around it was soft and yellow. Microscopically the tumour was a glio-sarcoma, with very thin-walled blood-vessels. (See Photographs, Plate 15.)

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A remarkable feature in this case is the long duration of the growth. Symptoms had been present for nine years before death occurred, and there can be no reasonable doubt that the tumour had been growing for at least that period. The slowness of the growth explains why the cardinal signs of intra-cranial tumour were so long delayed. Headache, vomiting, and optic neuritis are probably not caused by a cerebral tumour *per se*; they are directly dependent upon the rise of intra-cranial pressure, which is usually caused by such a tumour; they are always more marked and occur earlier in the rapidly growing tumours, which either add to the intra-cranial contents without merely destroying and replacing the tissues around, or which by



Serial coronal sections of the brain of A. B. The uppermost section passes through the ascending parietal convolution, the lowest through the posterior parietal.





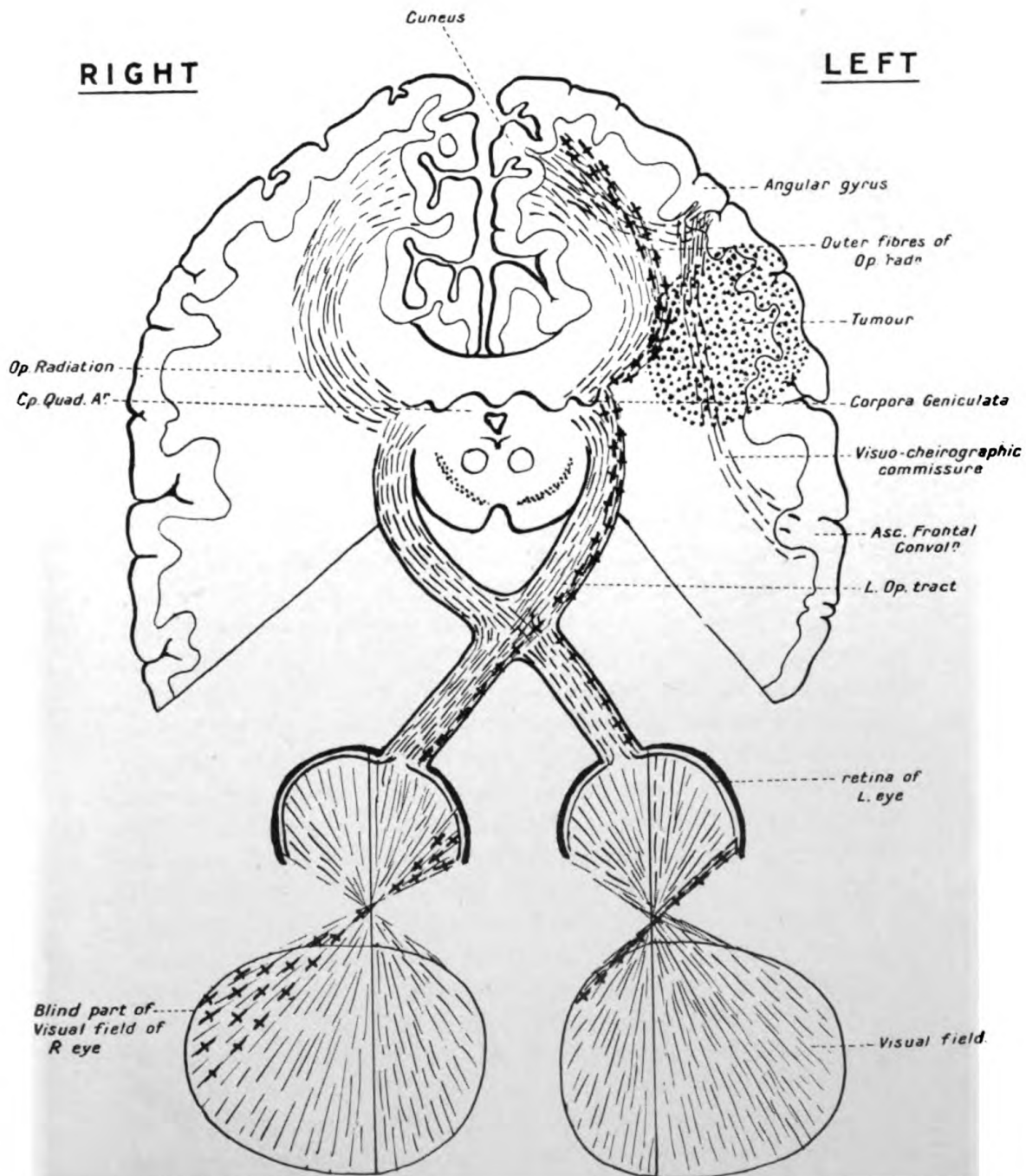
reason of their position give rise to a secondary hydrocephalus. Such a tumour constantly increases the intra-cranial tension; but if as in the patient described above, the tumour grows very slowly, sufficient time is allowed at each stage of the growth for compensation to occur, so that the intra-cranial tension is not so greatly raised as to give rise to the pressure signs. If, however, the compensation is disturbed by some such accident as a hæmorrhage into the growth; or if a tumour, latent or slowly growing, should take on rapid growth; then headache, vomiting, and optic neuritis are likely to occur. This is what appears to have happened in the above case, the hæmorrhage into the anterior part of the tumour having probably occurred less than a fortnight before death. It sometimes happens, especially in old people, that thrombosis of vessels running in or near a cerebral tumour occurs; in these cases, although the local symptoms may suggest rapid growth, there is no consequent increase in the intra-cranial contents, and such patients frequently die without having developed optic neuritis at all. It is quite possible that this may have been the cause of death in the father of this patient.

The absence of optic neuritis and severe headache made the diagnosis difficult in an early stage, and it was thought that the symptoms might be due to a syphilitic focus (vascular or gummatous) on the left Sylvian artery or one of its branches. The partial recovery under a course of potassium iodide favoured this diagnosis, and it was only during the last few weeks of life that it was felt certain that a cerebral tumour was present. That a sarcoma should have ceased for a time to cause progressive symptoms, and that general improvement should have occurred under potassium iodide is rather remarkable, although several similar instances have been recorded; probably the iodide acts largely by reducing the blood-pressure in general and therefore in the tumour, and so causes a general relaxation of the intra-cranial tension.

Not the least interesting point was the localisation of the lesion. Clearly the growth did not occupy at first any of the areas connected with speech, etc., for it was only when the diseased focus gave rise to an epileptiform discharge that there came on transient aphasia. At a later stage, when the patient first came under observation, the most marked feature was his

difficulty in writing. At this time he could understand speech perfectly, and could speak with only slight hesitation; indeed, when after a few weeks under observation he had recovered as far as he ever did recover, he could speak, read aloud, and understand speech and writing almost perfectly; then his only difficulties consisted in a partial degree of right hemianopsia, uncertainty in naming uncommon objects, and in his great defect of writing. Spontaneous writing, writing from dictation and copying, were all about equally affected; he once said that in writing from dictation he "could see in his mind's eye what he wanted to write, but he could not transfer the image to paper." Seeing that apart from his visual field difficulty he was able to understand written words perfectly, it was clear that the visual word-centre was intact; nor was there any evidence of involvement of the cortical representation of the ordinary movements of the hand, for these movements were all performed dexterously and powerfully—all except those of writing.

It has often been affirmed and almost as frequently denied that a special centre for writing exists; up to the present there has been reported no case of pure and complete agraphia. The famous cases of Pitres were incomplete in that the patients could still copy, and as Bastian points out they are best explained on the basis of destruction of the audito-visual commissure, leaving the visual and cheirographic centres and their connecting commissure intact. I do not wish to follow out in detail the discussion upon the question as to whether a cheiro-kinæsthetic centre exists; it will be remembered that Bastian concludes that though no actual proof has yet been given, such a centre exists as certainly as does the centre for articulate speech and in the corresponding hemisphere; that like the latter it is distinguished from the corresponding centre for the movements of the organ it supplies physiologically, although the various groups of cells and fibres subserving the two sets of functions may be anatomically blended. I think this case tends to support Bastian's theory; for a tumour was found occupying a position which, whilst leaving each of the word-centres intact and not involving the Rolandic cortex, must yet have cut some of the commissural fibres between these centres. Assuming that the fibres pursue a moderately straight course, the fibres coming forward from the visual word-centre to a possible cheiro-kinæsthetic centre in or near the Rolandic region



Diagrammatic horizontal section, to show position of tumour and fibres cut. Fibres corresponding to the blind area of the visual field are marked with crosses.



would be severely injured, as would those to Broca's convolution, whilst those connecting the visual word-centre to the auditory word-centre would be only partly severed, and the cheiro-auditory commissure would equally be injured but not destroyed. A lesion of this kind would fully explain the symptoms; seeing that the visuo-graphic and audito-graphic commissures are partly cut, only imperfect impressions would reach the cheiro-kinæsthetic centre, so that all forms of writing—spontaneous, from dictation and copying—would be about equally impaired, whilst the slighter injury to the visuo-auditory commissure would account for the occasional hesitation in reading aloud and for his slower understanding of written speech.

One other localising symptom was his partial right hemianopia. This was clearly due to involvement of the fibres of the optic radiation as they sweep round from the cuneus towards the thalamic region; evidently this group of fibres was only partly invaded, so that we are justified in concluding that in the region of the angular gyrus the most superficial (external) of the fibres of Gratiolet's radiation are those corresponding to the outermost part of the opposite visual field (see diagram, Plate 16).

In another case which has recently come under my observation the patient had been shot through the head in such a way as to produce a right hemiplegia (partial) and a hemianopia affecting his field of vision in both eyes below the horizontal level. The marks of the bullet wound were easily identified; their position was carefully noted and transferred to an excised hardened brain, which was transfixed so as to imitate the bullet wound of the living patient. It was then found that on both sides the optic radiation of Gratiolet was partly cut, the upper fibres alone having been severed.

It would therefore appear that the fibres of the optic radiation maintain their relative position throughout; that the uppermost fibres correspond to the lowest parts of the visual fields, and that the outermost correspond to the outermost region of the visual field on the opposite side.

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## Abstracts

### PHYSIOLOGY.

**ON THE PHYSIOLOGY OF THE ORBITAL LOBE.** FERRANNINI,  
(279) *La Rif. Med.*, 1903, No. 21 and 22.

THE author gives a long resumé of the experimental work which has been done in order to decide the situation of the olfactory centres in the brain, the results of which so far have been very conflicting.

He then gives details of his own experiments on nine dogs. He reached the brain through the temporal region of the skull, trans-fixed the frontal lobe, and separated the portion of cortex which corresponds to the orbital lobe.

The constant and persistent result of this operation was a marked disorder of the olfactory sense, hyposmia. This disturbance was found only on the side operated on, and was present for all olfactory sensations.

In a few cases taste was also disturbed, but only for a short time. This was probably due to a slight injury to the centre for taste which, according to Luciani and Seppilli, is situated in close proximity to the centre for smell. No other disorder of any special sense was noticed, but tactile and painful sensation appeared to be dulled, especially on the side of the body corresponding to the side of the operation. This was probably due to the shock of the operation. Often also a loss of strength was seen on the side of the body opposite to that operated on.

Bianchi also found paretic phenomena in the dogs in which he removed the frontal lobes, *i.e.* the portions of the brain directly continuous with the psycho-motor region.

Later, Dieulafoy has described a case of Jacksonian epilepsy which pointed to a tumour of the left Rolandic area, but in which, at the autopsy, a gumma was found implicating the tip of the frontal lobe and a part of the olfactory convolution. This may, according to Maragliano, have caused the motor disturbance. The author suggests that there exists in the orbital lobe some functional power which is capable of acting with that of the Rolandic area.

This is supported by the fact that bundles of degenerated fibres were found in the brains of all the dogs operated on, which started at the level of the wounded surface of the orbital lobe, and extended into the Rolandic convolutions, and ended in their lower middle third. A few degenerated fibres were traced into the lower

part of the superior third. These fibres are evidently fibres of association which have a long course.

No other bundles of degenerated fibres were found in the brain.

R. G. Rows.

## PSYCHOLOGY.

**THE DIRECTION OF INVESTIGATION FOR PSYCHOLOGICAL**  
(280) **WORKS.** W. WEYGANDT, *Centralbl. f. Nervenh. u. Psychiat.*,  
March 15, 1903, p. 176.

THE writer concludes his investigations on the above by an enquiry into the psychomotor mechanism employed in associative thinking in the acts of writing, muscular action and the problem of sleep; and devotes some space to researches in mental cases for similar acts. He employs a modification of the method of Aschaffenburg, which is to repeat a given word immediately it has been pronounced by the investigator and by means of a mechanism to record the time taken for the act. Weygandt makes the subject write the answer in shorthand in order to have less disturbance of association. A number of single syllable words are taken and combinations of them are used as test-words. The observations are conducted on the plan of Kraepelin's classification of direct and indirect associations. The average time for associating two syllable words was found to be 1.1 to 1.4 seconds; that of one-syllable words 0.9 to 1.2 seconds. The individual time was, however, very variable. The want of sleep lengthens the associating time considerably; drugs like trional have the same effect, but the author does not agree with the idea that the phenomenon is due to the circulation of waste products acting deleteriously on the brain. The effect of starvation was to prolong both the mental association and the physical, and to produce a diminution of the will power without much lessening of the comprehension.

The results of the administration of various stimulants and drugs are given, *e.g.* tea, caffeine, paraldehyde, chloral, morphine, chloroform, bromides, etc. The effect of the last-named was principally to cause a delay in committing words to memory; other mental acts were not materially affected. The observations on the insane were conducted on patients suffering from various mental affections, the results of which are given.

Paranoiacs and the subjects of chronic alcoholism could not be relied upon to give trustworthy results. In cases of dementia, the comprehension showed an average error of 13 per cent. Mispronunciation of words was usual, while the response to optical

stimuli was delayed. Memory faults were marked and showed an average of 50 per cent.

The subjects of mania with depression, during the stage of excitement, do not afford reliable data. An arrest of their attention sufficiently long for the experiments to be made is impossible. During the period of depression the association time was shown to be considerably lengthened. The writing peculiarities of such cases are described; the heavy, slow, laboured letter formation of the stuporose period is in marked contrast to the quick, badly formed, angular, faulty writing during excitement.

In epilepsy the results were not sufficient to found conclusions upon, but the difficulty of comprehension and the loss of will power are similar to the effects of alcoholism. Interesting and important data are given regarding traumatic neuroses, *e.g.* produced by railway accidents. The general findings were that there was a considerable increase in the number of mistakes in adding and subtracting numbers. Fatigue was also more readily induced.

W. MAULE SMITH.

### CLINICAL NEUROLOGY.

**PRIMARY FACIAL SPASM WITH CONTINUOUS FIBRILLARY (281) MOVEMENTS (MYOKYMY LIMITED TO THE DISTRIBUTION OF THE FACIAL NERVE).** H. FRENKEL, *Rev. Neurolog.*, June 30, 1903, p. 609.

A YEAR ago, Bernhardt recorded a case of primary tonic spasm of the muscles innervated by the left facial nerve, with constant fibrillary movements in some of the muscles which were the seat of spasm. A similar case was recorded more recently by Newmark, and Frenkel now records a third case. The patient was a married woman, age 22, who showed tonic contracture of the muscles supplied by the left facial nerve with constant fine fibrillary movement in some of the affected muscles, especially in the lower part of the orbicularis palpebrarum and to a less extent in the zygomatic muscle. Unlike the cases of Bernhardt and Newmark, Frenkel's case showed some changes in the excitability of the affected nerve and muscles—increased mechanical excitability of the muscles, slight diminution of faradic excitability of the muscles, and more so of the lower facial nerve, with no qualitative changes on stimulation with galvanism. The patient had never suffered from facial paralysis; none of the other cranial nerves were affected; there were no pains or painful points or affection of sensation or motion; no hysterical stigmata could be found.



As regards the ætiology of this condition, little can be said. In all three cases hysteria could easily be excluded—all three were women. Bernhardt's patient was subject to migraine; Newmark's patient was exposed to a chill and died some years later with symptoms of disseminated sclerosis. In Frenkel's case there was a history of some rheumatic pains, probably of little importance. She had carious teeth, but as they were as much on the right side as on the left, they were probably not the cause of the unilateral facial affection. At the time that this affection appeared the patient was under treatment for a deep keratitis and iritis, which yielded only to specific treatment of four months' duration, and the question naturally arises whether the cause of this eye condition was not also the cause of the facial spasm. This seems still more probable as the result of treatment with intramuscular injections of benzoate of mercury—after a few injections the facial spasms and later the fibrillary movements had almost disappeared.

A. W. MACKINTOSH.

**TABES AND MUSCULAR ATROPHY.** J. COLLINS, *Journ. Nerv. (282) and Ment. Dis.*, 1903, p. 324.

In tabetics slight localised muscular atrophy may lead to club-foot or other deformities; there are, however, severer forms occasionally met with in tabes which may resemble that of progressive muscular atrophy, and even produce severe motor paralysis. These amyotrophies have been described as a co-existence of tabes and progressive muscular atrophy, or as dependent on nerve changes. The latter is the explanation Collins adopts.

His case commenced with numbness and weakness, and with wasting of the small muscles of both hands at the age of 43. The wasting extended later to the forearms, and simultaneously difficulty in micturition, sexual impotence, and affection of walking appeared.

Eighteen months after the first commencement the patient noticed weakness of the one leg and some foot-drop, and six months later the other leg was similarly affected. The wasting progressed till he came under observation five years after its commencement. There was then marked wasting of the lower extremities, of the muscles of the pelvis, and of the hands. There was some fibrillary twitching and partial reaction of degeneration in the affected muscles.

Typical symptoms of tabes were present. Two years later he died suddenly with attacks of difficulty in breathing.

Besides the tabetic changes in the dorsal columns of the cord,

there was also found some degeneration of the lateral columns, especially in the lumbar region, but there was no definite pathological change visible in the cells of the ventral horns or of the spinal ganglia. The peripheral nerves examined had undergone parenchymatous and interstitial changes, and fibres of the wasted muscles were atrophied, with increase of their interfascicular connective tissue.

In this case, in spite of the marked muscular wasting, there were few subjective symptoms of tabes. Two somewhat similar cases of muscular atrophy in tabes are referred to. In the one the upper extremities were greatly wasted, probably due to previous alcoholic neuritis; in the other the shoulder muscles of one side were markedly atrophic without there being any visible cause for the wasting.

GORDON HOLMES.

**SYPHILITIC PSEUDOTABES. REPORT OF A CASE; THE  
(283) DIFFERENTIAL DIAGNOSIS OF TABES. JOSEPH COLLINS,  
*New York Med. Journ.*, April 4, 1903.**

THE patient was a labouring man, 47 years of age. He absolutely denied ever having had syphilis or other venereal disease, but had been "moderately alcoholic." The symptoms had begun ten years ago with shooting pains in the legs; progressive impotency, incontinence of urine and occasional loss of rectal control; difficulty in walking progressing to complete inability to stand, and diplopia were also complained of. The physical signs found were: static and locomotor ataxia, loss of knee and ankle jerks, hypalgesia, hypæsthesia, and especially ulnar anæsthesia, Argyll-Robertson pupils and hypotonia.

The patient died of acute Bright's disease; the post-mortem findings in the central nervous system alone are recorded.

The pia around the dorsal portion of the cord seemed to be somewhat thickened; the cord itself was a little shrunken and in the fresh state offered slightly increased resistance to the knife. Microscopically, the most remarkable feature throughout the mid-brain, pons, medulla and cord was a series of small hæmorrhages from the smaller vessels. The latter showed signs of extensive disease; many of them showed aneurismal swellings and much round-celled infiltration in and around their walls; large arteries in the fissures of the cord had enormously thickened walls, the tunica intima being especially involved. There was also present a large amount of small-celled infiltration in the white matter, particularly in the columns of Burdach, the infiltration being chiefly circumvascular. In the second dorsal segment one patch



was particularly obvious; it lay at the point of emergence of one of the posterior roots, and consisted of a fibro-cellular mass matting together the pia and supporting tissues of the cord at this point. In many places the newly formed tissue seemed to be on the point of breaking down. Stained by Weigert's method, sections from the mid-cervical region "appear when viewed under weak magnification to show slight degeneration of the posterior columns. . . . It is a question of interpretation whether or not it might be said that there is slight degeneration of a systemic character of the postero-internal columns in this region. My own conviction is that there is none." A certain amount of chromatolysis was present in the cells of Clarke's column and of the lateral horns.

The author concludes "that although there is nothing that is pathognomonic of exudative syphilis in the anatomical picture . . . the lesions are those that may be caused by syphilis, and in my opinion they are syphilitic." It is remarkable that the morbid changes described are so largely those which are usually associated with acute diseases, and it may be doubted whether the vascular changes were not associated with the acute renal disease which caused the patient's death; some of the figures show a condition closely resembling Wernicke's "polioencephalitis superior" which appears to be an acute or a sub-acute toxic process.

The author concludes with a discussion on the diagnosis of the syphilitic and parasymphilitic diseases of the spinal cord.

STANLEY BARNES.

**A CLINICAL REPORT OF NINE CASES OF FRIEDREICH'S  
(284) DISEASE; HEREDITARY OR FAMILY ATAXIA SO  
CALLED, WITH COMMENTS ON NOTEWORTHY  
SYMPTOMS. JOSEPH COLLINS, M.D., New York. Reprint  
from *American Medicine*, vol. v., May 1903.**

THE author details eight cases of Friedreich's disease which have been observed in his clinic during the last five years. The first part of the paper is devoted to a short account of the history of the disease and to a description of the chief features and clinical characteristics of the condition. He next describes the cases with comments. The following table (which I have drawn up from the reports of his cases) shows the uniformity of the clinical manifestations.

CASE	1	2	3	4*	5	6	7	8
Sex . . .	female	female	male	female	male	male	male	male
Age at onset .	13	10	10	15	9½	21	4	6½
Family affection.	3 out of 5	none	none	sister and	brother	one sister	Two brothers	
Pain . . .	dull aching in the back	pain in the feet	rheumatic pain in legs	stinging pain in legs	pain in knee	—	—	—
Pupils . .	normal	normal	normal	normal	normal	normal	normal	normal
Nystagmus .	absent	lateral	lateral	lateral	lateral	—	absent	lateral
Speech . .	unaffected	affected	affected	affected	affected	affected	affected	unaffected
Ataxia . .	marked	marked	marked	marked	marked	marked	marked	marked
	titubation	titubation	titubation	titubation	titubation	titubation	titubation	titubation
Knee-jerks .	absent	absent	absent	+	absent	on reinforcement	absent	absent
Ankle-jerks .	absent	absent	absent	normal	on reinforcement	absent	absent	absent
Plantar reflexes .	flexor	extensor	—	—	—	right extensor	flexor	right flexor, left extensor
Sphincters .	unaffected	unaffected	—	unaffected	—	—	unaffected	—
Sensation . .	unaffected	deep sensibility impaired	—	unaffected	—	unaffected	unaffected	deep sensibility affected
Deformities .	spine, club foot	spine, pes planus	spine, pes cavus	spine, manus cavus, pes cavus	spine	spine	spine, pes cavus	spine
Irregular Movements . .	—	present	—	—	—	—	—	present

\* This case showed the Ménière Symptom-Complex as well.

The uncommon features, pes planus in Case II., manus cavus, and the ménière symptom-complex in Case IV., in which the patient suffered from an indrawn and congested membrane, are all of interest. In discussing the presence of the knee-jerk in Case IV., Dr Collins cites many cases of Friedreich's disease in which the knee-jerks have been present, in some of which they disappeared later in the course of the disease. He would not make the presence of the knee-jerk any indication for placing a case in the category of Marie's hereditary cerebellar ataxia, which he does not regard as being a distinct disease. Mental weakness did not occur in any of Dr Collins' cases. He describes the ataxy of Friedreich's disease as differing from that of tabes, in that a reeling element is added to the gait, and that closure of the eyes does not increase the unsteadiness in the majority of cases. Titubation or the ataxia of the balancing muscles of the body is always marked. It is interesting to note that pain of various character occurred in five out of his eight cases; while irregular movements, a common text-book sign, were only observed in two.

T. GRAINGER STEWART.

**REMARKS ON PRIMARY NEUROTIC ATROPHY (OHAROOT-(285) MARIE-HOFFMAN TYPE) WITH REPORT OF A CASE IN WHICH THERE WAS EXCESSIVE INDULGENCE IN TEA AND COFFEE.** A. GORDON, *Journ. Nerv. and Ment. Dis.*, 1903, p. 354.

THERE is here published an aberrant form of muscular wasting to which excessive indulgence in tea and coffee are the only attributable aetiological factors.

A middle-aged man had for five years suffered with pain and a burning sensation in his toes, but only for a few months has there been any foot-drop or motor paresis.

On examination there was found some talipes equino-varus with marked foot-drop, the legs were emaciated but the calves were of fair size, and the muscles of the thighs and the small hand muscles were small and wasted. Some fibrillation was visible in the atrophied muscles, and reaction of degeneration was obtained in those more severely affected.

Besides the pain and numbness there was slight tactile loss in the feet, and the nails of the toes were atrophic. The knee and Achilles jerks were absent.

There was also some degree of optic atrophy and nystagmus on lateral deviation of the eyeballs. No family history, direct or collateral, of a similar condition could be obtained.

GORDON HOLMES

**THREE CASES OF PROGRESSIVE MUSCULAR DYSTROPHY (286) OCCURRING IN THE MALE MEMBERS OF A SINGLE FAMILY, AND COMMENCING AT THE SAME AGE IN EACH.** C. H. BUNTING, *Journ. Nerv. and Ment. Dis.*, 1903, p. 350.

THESE are three shortly reported cases of myopathy affecting all the surviving male members of a family of ten, but sparing the females. There was no family history of a similar condition.

At 5 years each boy began to get weak, and at 9 years of age each lost the power of walking. The condition of each on examination was very similar; marked wasting of the muscles of the shoulder girdles, some wasting of the upper arms, while in the lower extremities only the quadriceps extensor group was affected. There was no evidence and no history of muscular hypertrophy. The face of at least one case, the second boy, was weak and expressionless.

GORDON HOLMES.

**CONGENITAL SPASTIC RIGIDITY (LITTLE'S DISEASE) CAUSED  
(287) BY A FOCAL LESION OF THE SPINAL CORD OCCUR-  
RING IN INTRA-UTERINE LIFE. J. DEJERINE, *Rev.*  
*Neurol.*, June 30, 1903, p. 601.**

DEJERINE records a case of congenital spastic rigidity, proved post-mortem to be due not to cerebral disease, but to a focal lesion in the cervical region of the cord. The case was that of a man, age 63 years, born at full term, with spastic rigidity of all four limbs; marked contracture of all the limbs, rather more marked in the lower than in the upper limbs; no power of movement in the legs, and only slight power in the arms; rigidity of trunk and neck; all limbs normally formed; no true muscular atrophy, but slight diminution in size of the muscles of the arms; no affection of sensation or of sphincters; no implication of the face or of ocular muscles; tongue, pharynx, larynx intact; intelligence good; no epileptiform attacks. Death occurred at the age of 66 years. The autopsy showed no affection, macroscopic or microscopic, of cerebrum, cerebellum, pons-medulla. The spinal cord was a little smaller than normal, especially in the posterior part of the lateral columns. Microscopically, at the third cervical segment a focus of transverse sclerosis was found occupying the anterior two-thirds of the posterior columns, replacing the two posterior horns, and extending into each lateral column to terminate a little in front of the crossed pyramidal tracts. Above the third cervical segment there was a systemic sclerosis of the anterior parts of the posterior columns, terminating above at their respective nuclei. Below the third cervical segment there was sclerosis—or rather defective development—of the crossed pyramidal tracts, and also degeneration of the anterolateral descending tracts, which could be traced down to the first dorsal segment. In the focus of sclerosis there was a dense proliferation of the neuroglia fibres, and the vessels showed marked changes—thickening of the tunica intima, with reduction and, in some cases, complete obliteration of lumen, marked hypertrophy of muscular coat, thickening and great dilatation of lymphatic sheath, which contained a large number of granular bodies. At the level of the sclerosis the grey matter of the anterior horns showed diminution in the number of the cells, increase in the number of neuroglia fibres and thickening of the walls of the vessels.

Notes are also given of a case already recorded by the author (1897), in which the clinical picture and the pathological findings were very similar. The case was that of a man, aged 44 years, born at full term, who showed spastic paralysis of all limbs, much more marked in the lower limbs, and affecting the left side more than the right. The face, ocular muscles, tongue, pharynx, speech,

sensation, sphincters were intact. There was no history of fits, and the intelligence was fair. At the autopsy the brain was found intact, while in the cord, at the second cervical segment, there was a focal lesion similar in situation to that found in the first case, although not so extensive, and showing identically the same histological changes. Below this level there was sclerosis of the crossed pyramidal tracts, but no degeneration in the descending anterolateral tract. Above this level no degeneration in the posterior columns, but some degree of retrograde degeneration in the pyramidal tracts as far as the upper limit of the decussation.

Dejerine considers that these two observations establish the existence of a form of Little's disease due to a primary spinal lesion—a transverse myelitis—developed during intra-uterine life, and he believes that the transverse sclerosis found in both cases was the result of specific disease of the cord—the pathological changes being identical with those found in old-standing cases of transverse syphilitic myelitis.

Further, he thinks it may be possible *intra vitam* to differentiate this spinal form from the cerebral form. The spinal form should be suspected in a case of Little's disease, where the upper limbs are markedly involved without affection of the intelligence and of the facial muscles, and with no history of epileptiform convulsions. All these symptoms, it is true, are not infrequently absent in cases of Little's disease of cerebral origin, but only in cases in which the arms are little involved, or not at all.

In the discussion which followed the reading of Dejerine's paper before the Société de Neurologie of Paris, Brissaud and Pierre Marie objected to the inclusion of such cases as those reported by Dejerine under the name of "Little's Disease." Confusion had already been caused by a too wide application of the term, so as to include all forms of congenital spastic paralysis, and it was desirable to restrict the term to a particular form. Brissaud advocated its limitation to the *generalised* form of spastic rigidity, generally following premature birth. Objection was also taken to making the absence of epilepsy a diagnostic point in favour of spinal lesion—cases of the cerebral form, with pronounced rigidity, occurred without any epileptiform attacks.

A. W. MACKINTOSH.

**CONGENITAL ANTERIOR DISLOCATION OF THE KNEES, WITH  
(288) MALFORMATION OF THE ARTICULAR SURFACES; ITS  
RELATION TO LITTLE'S DISEASE. VINCENT, *Lyon*  
*Médical*, April 19, 1903.**

In the subjects of this congenital malformation the knees are hyperextended at birth instead of being flexed. The femoral con-

dyles are displaced backwards without altogether leaving the tibial articular surfaces, as though the interarticular ligaments were absent or unduly lax. While it is possible forcibly to overcome the contraction of the quadriceps and flex the knee, the articular surfaces of the joint cannot be manipulated into the normal position, and the limb, when left to itself, at once reverts to its former hyper-extended attitude, the leg forming a more or less obtuse angle with the thigh. Any stimulus of the skin increases the extensor spasm to a painful degree, and while the spastic condition is diminished in sleep, it at once occurs when the patient wakes. The infants are usually premature, and ultimately show signs of arrested mental development and other abnormalities; club hand and hyperextension of the elbows may be present. The condition requires tenotomy, which should be postponed till the second year, followed by retention of the limbs in the flexed position, and later, suitable gymnastic exercises.

Such cases must not be mistaken for Little's Disease, in which there is no alteration of the articular surfaces of joints or displacement of bones, and in which the adductors of the thigh and extensors of the foot are the muscles most affected, resulting in curving of the legs and talipes varus. While both diseases are doubtless due to an arrest of development of the cerebro-spinal axis, the absence of post-mortem observations does not permit of any opinion being formed as to the seat of lesion in the cases under consideration.

Short notes are given of the seven cases which Vincent has met with, on which the paper is based.

J. S. FOWLER.

**ON A CASE OF SPINAL TUMOUR (DORSO-LUMBAR REGION).**  
(289) F. RAYMOND, *Journ. de Neurolog.*, June 1903.

THIS article contains an account of a case which was used by Professor Raymond as the basis of a lecture on the above subject. The patient was a woman, aged 42 (the age is given later as 31), who in March 1901 began to experience constricting pain in the left iliac region. Her past history threw no light upon the condition. One of her two children had died at the age of 5 of tubercular meningitis, but there was nothing else suggestive of either tubercle or syphilis in her history.

The pain was at first intermittent, and only came on after prolonged walking; but in a few weeks the pain became very acute and radiated into the left thigh, frequently provoking attacks of hysteria. In October 1901 weakness of the left lower extremity came on, quickly followed by difficulty of micturition and defæcation and by weakness of the right leg.



When she first came under observation in the following January she was still well nourished and her only cranio-bulbar sign was a fine nystagmus of both eyes. The right lower extremity was rigidly extended and the left was held in a flexed position. Both had completely lost voluntary power and were very rigid; the feet were in the spastic position. There was anæsthesia to all forms of sensation up to a level half-way from the umbilicus to the pubes, the upper limit being ill-defined. There were no electrical changes in the muscles of the legs. There was no angular curvature of the spine, but the third lumbar spine was painful on pressure. There was a slight scoliosis with the concavity to the left. A short time afterwards it is noted that there is a very tender spot at the level of the second lumbar vertebra, and that the lower abdominal muscles were partly paralysed.

In his discussion of this case there occurs this paragraph: "Still later, with the painful paroxysms of the onset, so remarkable by their violence, was associated a state of motor paresis of the lower extremities. It observed the same order of invasion; the left side first, then the right. In all probability it was the extension of the compression to the anterior roots which take their origin from the lumbar region." It is difficult to understand the meaning of the last clause of the paragraph, especially as Raymond has described a condition of rigid paraplegia with greatly increased tendon jerks, and later reiterates that the wasting of the lower extremities was such as could be accounted for by "functional inertia" (p. 252). In discussing the nature of the lesion, he excludes spinal caries mainly on the grounds that it is rare to find it in a patient over 40 years of age, that it occurs almost exclusively in strenuous subjects who present other local signs of tuberculosis.

Raymond concludes that the case is one of non-malignant tumour within the spinal canal compressing the cord at the point of exit of the twelfth dorsal nerve-roots.

No confirmation either by operation or by post-mortem is recorded.  
STANLEY BARNES.

**A CASE OF TUMOUR OF THE AXIS ILLUSTRATING THE  
(290) FUNCTIONS OF THE THIRD CERVICAL SEGMENT.**

THORBURN and GARDNER, *Brain*, Spring, 1903, p. 120.

THIS case is one of sarcoma involving the left side of the axis, pressing upon the spinal cord between the second and third cervical roots of the left side and producing very complete paraplegia. It presents two special points of interest illustrating the surgical treatment of tumours in a very high level of the

spinal cord, and throwing light upon the sensory distribution of the higher cervical roots which are seldom observable in cord lesions.

A man, aged 51, began to suffer in 1897 from pain in the left shoulder and occiput. Paraplegia and anæsthesia developed very gradually in the following four years. In 1901 paralysis included all the limbs, the intercostal muscles and apparently the diaphragm, respiration appeared to be carried on solely by the accessory muscles and especially by the sterno-mastoids. Anæsthesia and

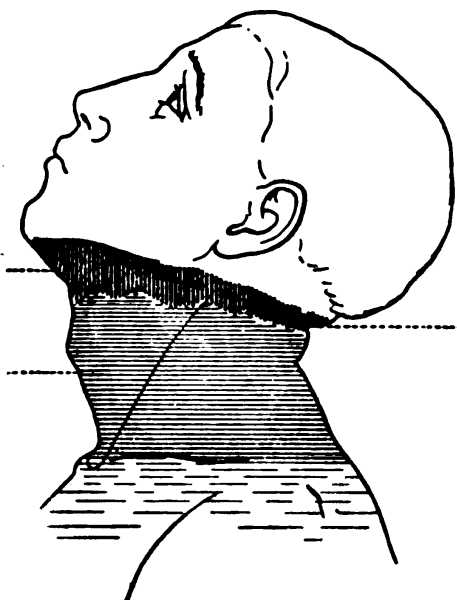
hyperæsthesia on the left side extended to the limits of the annexed diagram, but were less sharply defined and reached a lower level on the right side.

The patient being *in extremis* the laminae of the second and third cervical vertebrae were removed and a growth excised from the left side of the body of the axis. Marked improvement followed, but death ensued from gradual exhaustion some ten weeks after the operation. So far as the latter is concerned the writers find only one other case of excision of a tumour from so high a level.

The sensory distribution of the cervical roots is fully dis-

cussed, and reasons are given for the conclusion that the upper level of the third cervical territory is illustrated by the boundary of the anæsthesia in this case, viz., a line running almost circularly round the arch from the spine of the axis to the hyoid bone. The first cervical nerve having little or no sensory distribution the second root is defined by exclusion as filling up the area between this line and the fifth cranial nerve, "thus completing the localisation of the segmental distribution of the spinal nerves throughout the body." Numerous diagrams illustrate the views of previous observers, arrived at by other methods, but emphasis is laid upon the importance of not placing too much confidence in a single case, although similar examples of long survival after phrenic paralysis are likely to remain few in number.

AUTHOR'S ABSTRACT.



**SUCCESSFUL REMOVAL OF A CHONDROSARCOMA OF THE  
(291) SIXTH DORSAL VERTEBRA. ISRAEL, *Berl. klin. Wchnschr.*,  
1903, No. 22.**

ISRAEL records a case of successful removal of a tumour of the sixth dorsal vertebra and the cure of the paraplegia caused by the tumour. The patient was a healthy multipara thirty-nine years old. The first symptom was pain in the right upper quadrant of the abdomen. After this had continued for fifteen months weakness of the right leg appeared, followed some weeks later by similar weakness in the left leg. Three months after the first signs of paresis there was complete paralysis of both legs and of the muscles of the buttocks; also complete loss of sensation below the costal margins. The knee-jerks were exaggerated, ankle clonus and Babinski's sign were present, micturition was difficult. There was no deformity in the spine and no pain on percussion or pressure. The sequence of symptoms and the absence of degenerative muscle atrophy excluded a myelitis. Tumour of the cord itself was excluded because the symptoms had been those of sensory root irritation only for many months, followed by a hemiparesis on the side of the root irritation, and later by an extension of the paresis to the other side.

The symptoms were therefore attributed to an extra-medullary process. Syphilis and tubercle were considered as extremely unlikely, and a diagnosis of extra-medullary tumour was finally made. The sensory symptoms showed that the upper limit of the compression was at the level of the seventh dorsal segment. At the operation the arches of the fifth, sixth, seventh and eighth dorsal vertebræ were removed and a tumour three centimetres long was found growing from the body of the sixth vertebra. The tumour had flattened the cord and pushed it over to the left. Being very friable it had to be removed piecemeal by the sharp spoon and was found to have invaded the body of the vertebra extensively. After its removal the dura expanded and regained its normal appearance. Eight months after the operation the patient had almost completely recovered from all her symptoms. On examination the tumour was found to be a chondro-sarcoma, and the ultimate prognosis was therefore not favourable.

J. W. STRUTHERS.

**THE RELATIONS OF TETANY TO EPILEPSY AND HYSTERIA,  
(292) TOGETHER WITH A COMMUNICATION ON A CASE OF  
TETANY OCCURRING IN OSTEOMALACIA. ERNST  
FREUND, *Deutsches Archiv f. klin. Med.*, Bd. 76, H. 1-3, S. 10.**

AFTER briefly referring to the very important work done on the subject of tetany by Trousseau, Erb, Chvostek, Küssmaul, N. Weiss,

Frankl-Hochwart and Hoffmann, the author gives a short list of the commoner conditions in which the disease may occur (*e.g.* in certain diseases of children, in some gastric and intestinal lesions, in insufficiency or following removal of the thyroid gland, as a complication of pregnancy or lactation, various toxic conditions, infectious and nervous diseases), and then proceeds to discuss more especially its relations to epilepsy and hysteria, giving the following cases:—

CASE I. Female, *æt.* 28, wife of workman. Had three children, and on each occasion suffered from convulsions during lactation at periods of one year, eight weeks, and four weeks respectively after labour. After the first confinement there were only "slight attacks" in the extremities; after the second she had what appeared to be a typical epileptic fit (with convulsions, biting of the tongue, etc.); and on the third occasion, a fortnight after confinement, she had while in hospital an attack which affected the extremities, and which closely resembled tetany in character, and was followed later by a typical epileptic seizure, and also by an acute maniacal attack. Chvostek's facial phenomenon (contraction of facial muscles on tapping over seventh nerve) was present. Faradic excitability somewhat increased; galvanic greatly increased, and also very variable both at different times and on different sides of the body. Sometimes K.C.C., sometimes A.C.C. elicited first. The attacks of tetany followed the usual course, commencing with slight pain and tingling in the hands, followed by production of accoucheur's position, spread to lower limbs, etc. Contractions readily reproduced by pressure over the internal bicipital sulcus (Trousseau's symptom). The patient had several epileptiform convulsions at intervals, but no further spontaneous attacks of tetany; the latter condition however, according to the author, remaining latent, as the symptoms of Trousseau and Chvostek were demonstrable for several months afterwards.

CASE II. Schoolboy, *æt.* 11. Illness began three years previously "with a fright." Epileptic seizures (convulsions, loss of consciousness, cyanosis, foaming at mouth, etc.). On admission, well-developed boy; reflexes normal; galvanic excitability somewhat greater on right than on left side. During examination a tetanoid attack came on, and later, during his stay in hospital, he had convulsions, at one time epileptiform, at another tetanoid in character.

In these two cases the author makes the diagnosis of epilepsy and tetany, a combination of diseases which has already been described by several writers.

CASE III. Female, *æt.* 17. At the age of seven she became suddenly ill with convulsions and loss of consciousness lasting for 2½ hours, a condition diagnosed at the time as epilepsy. Her

mental development since this illness has been very backward. On admission, a pale, weakly, stupid girl. Chvostek's symptom moderately pronounced; Trousseau's phenomenon very marked, pressure inducing first a tingling sensation in the hand, followed by a typical attack of tetany. Faradic excitability not increased; galvanic much increased, A.O.C. and K.C.T. being obtainable with comparatively weak current, a condition however not specially marked in the facial nerve. On one occasion, during examination, pressure over the internal bicipital sulcus brought on a typical paroxysm of *grande hystérie* or hystero-epilepsy (convulsions, opisthotomus, passage of urine, retention of corneal and pupillary reflexes, etc.), and similar attacks afterwards occurred spontaneously. She also had a few tetanoid seizures.

CASE IV. Female, æt. 22, domestic servant. First attack of tetany three weeks after a confinement, and described as "commencing with a fright." Attacks recurred at frequent intervals, and during one of them there was loss of consciousness and vomiting. On examination, medium-sized, well-built girl. Pupils normal; conjunctival and corneal reflexes somewhat diminished; both knee-jerks increased; plantar reflexes, right much exaggerated, left lost, explained by the presence of total right-sided hemianæsthesia. On admission she had a typical paroxysm of hystero-epilepsy, in which she threw herself violently about. Her hands were held in the accoucheur's position, and the lower extremities were extended. Chvostek's symptom not specially pronounced; Trousseau's phenomenon present. There was a repetition of the hysterical attack a week later, after which Trousseau's symptom was strongly marked.

Tetany and hysteria are very seldom found in combination. Cases have been recorded by Westphal, H. Freund, Raymond and others, and the condition known as pseudo-tetany has also been described as occurring in conjunction with epilepsy. The author classifies Case III. as one of tetany and hysterical convulsions, while he thinks that in Case IV., also undoubtedly a case of hysteria, the diagnosis of tetany is probably correct, though not absolutely certain, as the condition, especially in the later stages, might have been one of pseudo-tetany.

He concludes that in the course of an attack of tetany paroxysms resembling epilepsy or hysteria may occur, not as a complication, but as the expression or outcome of a common pathological basis. The chance combination of tetany with one or other of these diseases may, and in fact does, sometimes occur; and again, epilepsy or hysteria may supervene in the course of certain forms of muscular cramps (pseudo-tetany), which might at first sight be mistaken for true tetany, but the latter can always be differentiated from these by careful clinical observation.

The author then gives a brief outline of a case of tetany occurring in conjunction with osteomalacia.

CASE.—Female, æt. 29. Admitted at beginning of November 1901. Married 10 years; no children nor abortions; menses commenced at 14, regular; have ceased for the last year. Her present illness began 2 years previously, gradually increasing to its present severity, and now presenting the typical clinical picture of osteomalacia. (Refers to description of this disease given by Hugo Beckmann, p. 5, same issue of Journal.)

The attack of tetany supervened in this case some 4 weeks after admission. On the first occasion there were spontaneous tonic contractions in the upper limbs, the hands being held in *main en griffe* position, each attack lasting about five minutes. Between these Trousseau's symptom was obtainable, and Chvostek's facial phenomenon was exceptionally active. A fortnight later the patient had a recurrence of tetany, and again at intervals for five or six months. Her general condition is now much improved, and she has had no further attacks since, nor are Trousseau's and Chvostek's symptoms now obtainable. The author claims that in this case the osteomalacia was, if not the direct, at all events the predisposing cause of the tetany, as none of the other usual causes of the disease could be made out.

W. E. CARNEGIE DICKSON.

**TIC IN HORSES AND TICS FROM IMITATION IN MAN.** RUDLER (293) et CHOMEL, *Rev. Neurol.*, juin 15, 1903, p. 541.

THERE is here presented an interesting study on what appears to be a fairly frequent variety of tic in horses—*tic de l'ours*. It consists in irregular movements of all four limbs and of the head and trunk which persist even during perfect rest, but increase under observation.

This condition seems to be almost invariably due to imitation of another horse similarly affected, and in a large stable the animals which become interested in, and finally develop the tic movements of a fellow generally show evidence of an abnormal psychical state, nervousness, restlessness, irritability, occasionally vicious habits, and in many definite stigmata of degeneration as corporeal asymmetry were present.

In development the movements are first conscious and voluntary, adapted to a definite aim—imitation of similar movements; then echokinesia movements only partially voluntary, which occur only in the presence of the animal originally mimicked. Till this stage cure by isolation is possible, and if not adopted the acts, by repetition, become automatic, are unconsciously reproduced, and con-

tinue to be so without further cause or aim and thus constitute a typical tic.

This explanation is that of Meige and Feindel of the origin of tic in man; that a purposeful movement, the result of either external or primarily cortical stimulation, finally becomes, in suitable subjects, habitual and automatic, and is then to be regarded as a psychomotor affection independent of will or consciousness.

GORDON HOLMES.

**ON KORSAKOW'S SYMPTOM-COMPLEX** (Zur Lehre vom Korsakow'schen Symptomencomplex). E. MEYER and J. RAECKE (of Kiel), *Archiv f. Psych. u. Nervenl.*, Bd. 37, Heft 1.

THE authors give a careful analysis of eight cases presenting Korsakow's syndrome—enfeebled attention, loss of orientation, confabulation—the course, ætiology and differential diagnosis being specially discussed. They agree with most authorities that polyneuritis is not constantly present. The psychosis is due to some serious organic lesion of the central nervous system. In their cases the causes were general paralysis, dementia postapoplectica, brain tumour, chronic alcoholism with pupil phenomena and loss of knee-jerk. Complete recovery never occurs; the pathological anatomy gives no general results. The syndrome is not a disease *sui generis*, and is certainly not an exclusively alcoholic psychosis; this is contrary to the view of Bonhoeffer, who regards it as a chronic delirium. Bonhoeffer is not justified in separating a form with initial delirium tremens as the true Korsakow's syndrome. The authors call attention to the great difficulty of diagnosis from general paralysis when the syndrome is associated with loss of pupil reaction and of knee-jerk, and with a history of chronic alcoholism.

C. MACFIE CAMPBELL.

**THE LOCALISING IMPORT OF THE TICKLING-REFLEX IN (295) CEREBRAL AFFECTIONS.** A. PICK, *Wien. klin. Wochens.*, H. 13, 1903.

THE writer considers that too little importance has been attached to loss of the tickling-reflex, *i.e.* the involuntary laughter produced by tickling the armpits and soles, and he records a case to show its value in localisation.

The patient, a merchant aged 37, of neurotic family history, and with a probability of syphilis, had five apoplectic seizures in the end of 1888, within a few days, in the last of which he lost his speech, power of right side, and to a certain degree his memory. In 1899 the hemiplegia was marked, with contractures of right arm and leg, blunting of cutaneous sensibility on the right side, and deterioration of the mental condition. The reflexes were then all

present, and in general more vigorous on the left side, but what was particularly observed during the whole period from 1891 was that *while on the left side tickling of the soles and especially of the armpits produced the normal effect, there was no response to tickling on the right side.*

He died in May 1900 from double pneumonia. The points of importance in the section were:—Macroscopically a suppurative leptomeningitis and atrophy of the whole brain. Microscopically the spinal cord showed the expected degeneration of the right-crossed pyramidal tract, and of isolated fibres in the left; in the medulla, degeneration of the left pyramid, traceable upwards to a centre of very old standing softening in the innermost part of the lenticular nucleus and superjacent part of the internal capsule; with other minute areas further back in the lenticular nucleus and more mesially in the internal capsule.

Brissaud and v. Bechterew have established with great certainty the optic thalamus as the centre of the tickling-reflex, but the fibres connected therewith are in doubt. The writer believes that in this case the loss of reflex is accounted for by interference with these fibres. To the objection that softenings are not uncommon in this region, he answers that the presence of the tickling-reflex has obtained very little attention, while the loss of reflex combined with the slight impairment of sensation he attributes to the very slight interference with the sensory paths.

Only one case could be found, by the writer, of a similar observation, and this (*Bleuler Archiv. f. Psychiatrie*, 25) he quotes as confirmatory.

JOHN D. COMRIE.

#### **HYPERÆSTHESIA OF THE NAILS (ONYCHALGIA NERVOSA).**

(296) H. OPPENHEIM, *Monatsschr. f. Psychat. u. Neurol.*, Bd. 13, 1903, S. 265.

THE writer draws attention by the record of three cases to a condition which has received no particular notice, but which may develop in persons of neuropathic diathesis in association with neurasthenia or hysteria.

Z., aged 36, official, of nervous family, suffered as a child from night terrors and headaches, later from nervous dyspepsia, and recently from worry and insomnia. Since early childhood cutting and brushing the nails, even pressure on the nail-bed, have given him pain, and for a whole day after cutting the nails the hands are useless owing to the pain of pressure on the finger points.

F., aged 12, a boy, suffered, like his mother, from typical migraine. From earliest childhood he resisted to the utmost the cutting of his nails, and after cutting remained several hours with widespread fingers unable to bear a touch upon the tips.

S., aged 46, a woman of neurotic family, suffered from flatu-



lence, globus hystericus, low spirits, and sleeplessness. After three small accidents to the finger nails these became so tender that to play piano, draw on gloves, or even to grasp objects firmly became impossible, and in her case also careless cutting or brushing of the nails occasions much pain.

The only records of similar cases discoverable by the writer are one by Joseph, a case of erythromelalgia by Gerhardt, and a reference in Hebbel's autobiography.

JOHN D. COMBIE.

**A CASE OF MERALGIA PARESTHETICA (BERNHARDT'S (297) SENSIBILITÄTS-STÖRUNG), WITH A SHORT ACCOUNT OF THE CONDITION.** EDWIN BRAMWELL, *Edin. Med. Journ.*, July 1903.

**MERALGIA PARESTHETICA** is here defined as "a clinical entity which is characterised by paræsthesia, and usually more or less objective disturbance of sensation of the skin of one or both thighs, confined to the area supplied by the external cutaneous nerve." The case here described is that of an army reservist, aged 43, who attributed his condition to sleeping in damp clothes on hard boards. Pain in the region of the right hip was a prominent symptom and was so severe as to prevent the patient working. On the outer side of the right thigh from the great trochanter downwards was an area where pin pricks were not distinctly felt as painful; over this area tactile sensation was slightly impaired, while the perception of both heat and cold was distinctly defective; in this region a Faradic current sufficiently strong to cause considerable pain elsewhere was well borne. There was marked tenderness over a spot which corresponded to the point of emergence of the external cutaneous nerve through the deep fascia. Rest in bed, Faradism and drug treatment having failed to produce any beneficial effect in the pain, Mr Alexis Thomson excised a piece of the external cutaneous nerve. This was followed by temporary disappearance of the pain, which remained absent for a month but then returned. The excised piece of nerve, which was examined by the methods of Weigert and Busch, appeared to be quite normal. The author has only been able to find one other case of this disorder in the literature in which a portion of the nerve was resected. In this case, which was reported by Souques, the excised portion of nerve also appeared to be quite healthy.

A brief record of the more important symptoms of the condition is appended.

AUTHOR'S ABSTRACT.

**THE MIMICRY OF GASTRIC TROUBLES BY SPINAL DISEASE.** (298) SIR FREDERICK TREVES, *Practitioner*, Jan. 1903, p. 1.

IN an interesting little paper, Sir Frederick Treves points out that unrecognised cases of spinal disease may present symptoms which

not uncommonly lead to an erroneous diagnosis of gastric disorder. Thus a child may suffer from persistent "bellyache," the abdominal pain depending actually upon involvement of spinal nerve roots secondary to vertebral caries. Two cases of what at first appeared to be dyspepsia are described. In the first, the symptoms apparently resulted from dorsal caries; in the second case, the abdomen was opened but nothing pathological found. A sarcoma growing from the mid-dorsal region of the spine was detected at a later date.

The author draws the following moral from these cases:—"Suspicion may be aroused as to the genuineness of a gastric trouble, when pain is the all-predominating symptom, when it is intense and persistent, and when vomiting is at the same time either absent or insignificant."

EDWIN BRAMWELL.

### PSYCHIATRY.

**BACTERIOLOGICAL AND CLINICAL OBSERVATIONS ON THE (299) BLOOD OF CASES SUFFERING FROM ACUTE CONTINUOUS MANIA.** LEWIS C. BRUCE, *Journ. Ment. Sc.*, April 1903.

THE author was unable to find any organisms in the blood of acute recent cases of insanity—except in one case of general paralysis.

Dr Bruce, acting upon the theory that any organisms present in the blood would reveal themselves if a suitable nidus was provided for their growth, produced a localised abscess in 25 patients suffering from various forms of insanity by injecting 2 c.c. of turpentine into the soft tissues of the flank. The abscess was aspirated on the third day, and a couple of drops of the fluid obtained added to tubes containing sterile nutrient broth. The tubes were incubated for 48 hours, and at the end of this period microscopic examination of the broth demonstrated the presence of a small diplo-bacillus in eight cases of acute continuous mania. A hanging drop culture showed that the bacillus grows in chains and clusters and that it is slightly motile; it holds Gram's feebly and does not take up the commoner dyes well. The bacillus is not fatal to rabbits, guinea-pigs, or white mice.

Dr Bruce failed to isolate the bacillus from the skin and faeces of patients suffering from acute insanity.

The abscess had no deleterious effect on the patients, and in cases of acute mania the beneficial effect was so marked that Dr Bruce never hesitates to induce an abscess in every case of acute mania which does not rapidly improve under ordinary treatment.

It is through the leucocyte action of the blood that nature effects a recovery in all cases of acute mania, and the formation of an abscess simulates and surpasses the leucocyte production which

naturally occurs when a patient recovers, and which even persists after recovery is completed.

Dr Bruce states that an examination of the blood in acute mania is a valuable aid to prognosis. He found that there is a high leucocytosis during the first few days of the disease, and the higher the leucocytosis and the higher the percentage of the polymorphonuclear cells the better is the prognosis; on the other hand, if a case of mania has lasted a month, remains maniacal and sleepless, and the blood examination gives a leucocytosis of 14,000 per c.mm. with a percentage of 60 or below 60 of the multinucleated cells, then the chances of an immediate or early recovery are poor.

In a case of acute mania which does not recover the leucocytosis tends to remain between 12,000 and 16,000 per c.mm., and after one or two years the polymorphonuclear cells may only average from 20 to 50 per cent.

The formation of an abscess in a case of chronic mania produces only a temporary leucocytosis.

These observations do not apply to the mania of "folie circulaire," nor to the mania of alcoholic poisoning.

H. de M. ALEXANDER.

**REVERSALS OF HABITUAL MOTIONS, BACKWARD PRO-  
(300) NUNCIATION OF WORDS, LIP WHISPERING OF THE  
INSANE, SUDDEN FAILURES OF VOLITION, REPETITION  
IMPULSES.** S. WEIR MITCHELL, *Journ. Nerv. and Ment.  
Dis.*, April 1903, p. 193.

THE series of hitherto undescribed mental phenomena, which the author calls "Reversals," are illustrated by several remarkable cases. The term "Reversals" is here defined in the author's words.

"These assumed two forms in the first case seen by me. The opposite of the thing willed was done, or else what it was meant to do was done in a way which reversed the usual manner of doing it."

The first case was that of a middle-aged naval officer, in every way healthy, but anxious-minded. After a period of active service he suddenly noticed that while he had ascended a flight of stairs he would find himself descending the stairs he had just ascended and generally going down backwards, also ascending and descending backwards, locking a door instead of unlocking it. When he observed himself doing these things he could check himself. "During the years of this peculiarity," says the author, "he served in the war with distinction."

The second case was one of a middle-aged lady who developed the habit of reading the end of a book before the beginning, which arose, as she says, "from an over-indulged curiosity to know how the book ended."

The third and fourth cases are somewhat alike. The former, a woman aged fifty, suffering from arterial degeneration, albuminuria, and attacks of mental disorder, who would put on her undervest over her feet, her drawers over her head, her shoes on her hands and her gloves on her feet; the latter an albuminuric, over-worked officer, who acted in practically the same manner.

The next case was that of a dentist who, while suffering from severe headaches, always said the reverse or negative of what he intended, and being conscious of the fact would refrain from speaking. The attacks occasionally ended in delirium and semi-coma. Change of climate and occupation effected a complete cure.

Another interesting case, that of a man who died of cancer of left anterior cerebral lobe, illustrates backward pronunciation. This man used to say "Tac-im" for "my cat," and "dog-ho" for "oh God," etc.

The author next deals with the common habit of lip and tongue movements during thought in people on the borderland of insanity, and describes an experiment of Dr Scriptures which registers the tongue movements during thought.

Then follows an account of obsessions, or as the author prefers to call them, "despotic habits," arising from asthenic mental states.

Counting steps, walking on alternate stones and such like trivial habits should be watched, he says, as in the end they may become masterful, and cites amongst others some of Dr Johnson's habits related in "Boswell's Life of Johnson." "When by long habit," says the author, "some mental process has been connected with a form of physical movement, to break the motion may interfere with an associated intellectual activity."

As an illustration of a variety of despotic habits in one patient, the author cites the history of a young woman who had an intense desire to kill her children, which only ceased at the age of fifty, when her obsessions changed completely; for instance, she would always look in her water-pitcher, otherwise there would be a snake in it; when making a call she would have to move a table or chair before leaving the house; she was afraid to look at an engine for fear she would throw herself underneath it, and so on. Her mind was quite clear and she was an efficient mother and housekeeper and a great reader.

In conclusion the author mentions a condition in which there is "a suddenly acquired incapacity to do, not a certain class of things, but one particular thing." Such cases are that of a lady who once in a year or two finds it impossible to answer some single letter, and another member of her family was similarly annoyed by finding himself unable to pack his portmanteau on a certain occasion.

As to obsessions in childhood, Dr Mitchell mentions two of his own brothers, one of whom as a child had a great dread of feathers, while the other would not go out of doors without an umbrella, a habit which lasted for some years from the age of ten.

T. GRAINGER STEWART.

**ON DEMENTIA DUE TO A PECULIAR FORM OF CORTICAL (301) ATROPHY.** M. PROBST, *Arch. f. Psychiat. u. Nervenkrank.* Bd. 36, 1903, S. 762.

THIS is a careful study of a case of acquired mental weakness, dependent on a peculiar form of cortical atrophy.

The patient was a woman, æt. 21 on admission, free from hereditary taint, and with previous good mental and bodily health. She was said, however, to have been addicted to alcohol. Married six weeks before admission, a month later she became careless and forgetful, and then gradually depressed and confused, with some stupor. On admission she appeared restless, was constantly smiling, and her conduct was silly. She was unable to do simple calculations correctly, and her replies were slow and contradictory, and she omitted or added letters or syllables in writing, but her memory was fair. Sleep and appetite were good. Faintness of left nasolabial fold, tremor of tongue and hands, and Romberg's phenomenon were the only physical signs. For about two years she was quiet, depressed and weak-minded, but from that time until her death, some thirteen years later, she was maniacal, restless, excited, idiotic, irritable, and sometimes aggressive. She was erotic and masturbated, was dirty in her habits, had an enormous appetite, and slept but little; and there were frequent periods of violent excitement. She became exceedingly fat. Contracture of the right toes developed, and at one period she had a series of epileptic seizures, in which the spasms were more marked on the right side. A uterine sarcoma was removed, but recurred locally in the occipital bone; death resulted, however, from pyelonephritis and purulent bronchitis. Towards the end her speech was hesitating, and she usually repeated what she said. Hearing was not noticeably affected, but there was probably some loss of tone-perception; sight was normal; knee-reflexes were exaggerated.

A very careful examination of the brain and cord (the former in uninterrupted serial sections) showed the following:—The membranes and vessels were practically normal throughout. Both hemispheres showed atrophy (the right much more than the left) in the fronto-parietal and temporal regions, atrophy being most marked about the island of Reil on both sides. The chief atrophied parts were: right, superior frontal, anterior and posterior central (upper part), and orbital part of first frontal convolutions, the whole

island and its operculum, the three temporal convolutions and the temporal pole, the fusiform and uncinate gyri, the supramarginal gyrus and gyrus fornicatus. On the left side the superior frontal convolution, orbital part of first frontal, island and operculum, first temporal, fusiform and uncinate gyri. Both amygdaloid nuclei were atrophied, and there was some atrophy of the right lenticular nucleus and optic thalamus. There was atrophy also of the white centre on the right side and especially of the outer and inner capsules; the anterior commissure, the fimbria, fornix, cornu ammonis, and olfactory tracts on both sides were small, also the right internal geniculate body, etc. A sarcomatous cyst penetrating into the right posterior horn, through the third temporal and fusiform gyri. The medulla oblongata and cerebellum were normal, and the cord also, except for recent degeneration of Goll's column.

The cortical atrophy was of unequal degree and irregularly distributed, often in isolated spots. It varied from a granular state of the large pyramidal cells to total disappearance of all ganglion cells, with the tangential fibres, and in the most advanced parts, of the underlying medulla, only alveolar neuroglia being left. The neuroglia merely showed slight compensatory hypertrophy as a rule, and there was never sclerosis. The process is judged to be a primary cortical atrophy, multiple and circumscribed, and of a peculiar kind.

W. R. DAWSON.

### TREATMENT.

**INTRACRANIAL NEURECTOMY.** HOWARD D. COLLINS, *Ann. Surg.*, (302) May 1903, p. 665.

COLLINS reports a case where extensive temporary paralysis followed an operation for avulsion of the right Gasserian ganglion. The patient had a markedly scaphocephalic skull, and in exposing the foramina of exit of the second and third divisions of the fifth nerve, the brain had to be crowded more towards the median line than usual. The second and third divisions of the nerve were divided, grasped in a pair of forceps, and as much of the ganglion tissue as possible, torn away. After the operation the patient became very drowsy and lethargic, and complete paralysis of the right third, fourth and sixth cranial nerves appeared. Partial paralysis also appeared in the left arm and leg. The paralysis gradually disappeared, and seven months after the operation the patient reported that he was as well as ever, though not quite up to his full strength.

Collins attributed the paralysis to pressure by the retractor in making a suitable exposure of the very deep middle fossa.

J. W. STRUTHERS.



**THE SURGICAL TREATMENT OF FACIAL PARALYSIS BY  
(303) NERVE ANASTOMOSIS. HARVEY CUSHING, *Ann. Surg.*, May  
1903, p. 641.**

ONE of the most successful of the few reported cases of spinal-accessory facial anastomosis is recorded by Cushing. The operation was undertaken for the relief of a complete facial paralysis, caused by a revolver wound of the right mastoid process. The bullet had carried away a portion of the nerve between the geniculate ganglion and the stylo-mastoid foramen. Six weeks after the injury, when the bullet wound had healed, the spinal-accessory nerve was exposed at its point of entry into the deep surface of the sterno-mastoid muscle, completely divided, and united to the distal end of the facial nerve, which was freshly divided just beyond the stylo-mastoid foramen.

Eighty-one days after the operation there was marked improvement in the patient's condition. Facial asymmetry at rest was hardly noticeable, voluntary motion was possible in the orbicularis palpebrarum, and to a less extent in the lower lip. Gradual improvement continued, and at the time of the last note, 287 days after the operation, the condition was as follows:—Volitional control of individual groups of muscles in the face had returned, and could be effected without associated shoulder movements or contraction in the other facial muscles. Emotional expression, however, had not improved in corresponding degree, and was still associated with considerable asymmetry. The electrical reactions on the side of operation were practically normal. Violent elevation of the shoulder caused contraction of the entire facial group of muscles, as did vigorous rotation of the head to the left. Motions of less vigour, however, were possible without producing any accompanying contraction of the muscles of expression.

The return of the power of disassociated facial movements in Cushing's case is interesting in view of the statement by Ballance and Purves Stewart after an experience of six cases of facio-accessory anastomosis, that "recovery appears to be limited to associated movements in conjunction with the shoulder."

Cushing makes two suggestions for the possible origin and course of impulses causing facial movements after his operation.

1. That the cortical centres concerned in shoulder movements (trapezius), and rotation of the head (sterno-mastoid), may themselves in the course of time be educated by training to co-ordinate the impulses, which have been side-tracked into the motor area of the facial nerve, so as ultimately to lead to expressional movement.
2. That the cortical centres originally presiding over movements of the face continue to play a part in the co-ordinate action of the facial muscles, possibly influencing the higher neurones of the N. accessorius through the intermediation of connecting tracts in the cortex.

J. W. STRUTHERS.

**ON THE DIETETIC TREATMENT OF EPILEPSY.** RUDOLPH (304) BALINT, *Neurol. Centralbl.*, April 16, 1903, p. 347.

BALINT quotes the satisfactory results obtained by Schäffer, Gerbini, Krell, Cappelletti and D'Ormen, Lyon, Hudovernig and Schnitzer, by withdrawing sodium chloride from the diet of epileptics. On the contrary, Hahnly and Bagerus observed no benefit in their cases. The author, whose previous results are confirmed by the former group, wished to determine the effect of prolonged treatment by a chlorine-poor diet. Weakness, alleged to occur during treatment, occurs only if the patient tires of the diet and eats little. The patient should be weighed weekly and the diet must be acceptable. At the outset all were dieted on milk, butter, egg, fruit and bread, in the preparation of which sod. bromide was used instead of sod. chloride. Patients treated at home frequently tired of the diet and then vegetables, flesh and farinaceous foods were allowed, sod. bromide as before replacing common salt for cooking purposes. If a seizure occurred, strict dieting was again resorted to. If the weight was diminished or bromism threatened, concessions of the diet were allowed. Five inveterate cases were treated in hospital. The average number of seizures per case per week before treatment was 8.2; during treatment continued for six months, 1.8. Seven cases were treated at home. These reported themselves weekly. Two were observed for 1½ years each. The results were equally satisfactory. For adults 3 grammes sod. bromide were given daily, for children 1 to 1½ grammes. If bromism threatened, this was diminished or omitted for a time. On the part of the doctor the treatment demands great patience; on the part of the patient and his household much intelligence and will power. The treatment is even carried out with difficulty in hospitals on account of the special preparation of the food required and also because careful guard has to be kept that the patient does not obtain other foods. Only in sanatoria for epileptics could preciseness of individual dietetic management be guaranteed. But results already obtained indicate, that it is practicable in less favourable circumstances with the necessary perseverance and intelligence and under medical guidance. The mental and bodily state of the patients almost invariably improved. J. EASON.

**THE RESULTS OF SURGICAL TREATMENT IN BRAIN TUMOURS.** M. ALLEN STARR, *Journ. Nerv. and Ment. Dis.*, July 1903, p. 398.

WE have here from the pen of Dr Allen Starr a statistical enquiry into the results of operations for the removal of brain tumours, together with an appendix by Dr O. Hensel consisting of 202 references to cases operated on and reported between the dates



January 1st, 1896, to January 1st, 1903. This bibliography, which is systematically arranged according to the results of operation and the region of brain involved by the new growth, will without doubt prove of the greatest value. Dr Starr has analysed 365 cases which have been operated on for removal of a brain tumour, including those collected by him and reported in 1896 and 1897. He divides these cases of tumour—315 of which were cerebral and 50 cerebellar—into four groups:—

- (a) Cases in which the tumour was not found (111).
- (b) Cases in which the tumour was found but not removed (27).
- (c) Cases in which the tumour was removed and the patient died (59).
- (d) Cases in which the tumour was removed and the patient recovered (168).

No definite statement is made as to the exact sense in which the term recovery is used, but on glancing over the bibliography we notice that a number of cases are included as recoveries in which death took place within a few weeks of the operation.

The above figures cannot, in our opinion, for one moment be taken as affording any indication of the chances of success in operations for the removal of cerebral tumours, and we are at one with the author when he states that "too great importance should never be attached to statistics" although "they possess a certain value and are never without interest." Possibly the day may come when 46 per cent. of cases carefully selected and operated on for the removal of a brain tumour may be classed as successes, but Dr Starr we are sure would be the first to agree with us when we say that such a percentage of successes gives an absolutely erroneous impression of the risks of the operation at the present day. The fallacy is accounted for by the circumstance that physicians and surgeons as a rule record only their successful cases.

Dr Starr points out that the presence of a brain tumour and the possibility of its being within reach of the surgeon can be much more readily decided now than formerly was the case. The rapidity with which the skull can be opened has increased the chances of successful operation, as has the recognition of the need for the removal of a considerable area of bone in these cases.

The operations for the removal of cerebellar tumours are, in Dr Starr's experience, attended with great difficulty and danger. He sums up his opinion on the operative treatment of cerebellar tumours by saying that "after a considerable experience it seems to me futile to attempt this operation."

The causes of failure in operations for the removal of brain tumours he ascribes to:—

- (a) Mistakes, sometimes unavoidable, in the localisation of the tumour.

(b) Inaccessibility of the tumour or impossibility of removing it owing to its infiltrating character; and lastly,

(c) The dangers of hæmorrhage and meningitis, which are fortunately less dreaded than formerly.

In this paper palliative operations for the symptoms of increased intracranial pressure, headache, optic neuritis, etc., are not referred to.

EDWIN BRAMWELL.

## Review

**THE NEURONE THEORY AND ITS ADHERENTS. A CONTRIBUTION TO THE SOLUTION OF THE PROBLEM OF THE RELATIONS BETWEEN NERVE CELL, FIBRE AND GRAY.** (Die Neuronenlehre und ihre Anhänger. Ein Beitrag zur Lösung des Problems der Beziehungen zwischen Nervenzelle, Faser und Grau). By FRANZ NISSL (of Heidelberg). With two plates.

THE Neurone theory was first suggested by His on the basis of his histogenetic work, and independently by Forel in view of Gudden's experiments and Golgi preparations. After Ramon y Cajal's work Waldeyer definitely formulated the theory: "The nervous system consists of numerous nervous units (neurones) which are anatomically and genetically independent: each neurone is composed of three parts—nerve cell, nerve fibre, terminal arborisation."

In 1898 Nissl showed that the theory was no longer tenable in view of the existence of a specific "nervous gray." "Die Neuronenlehre" is written in answer to his critics, and to give this doctrine its final quietus.

Some, while clinging to the doctrine, see that it is necessary to modify it. Edinger, Hoche, Münzner substitute for the anatomical unit of Waldeyer respectively a biological, a functional-trophic, a trophogenetic unit. Their theories are untenable, for the concept is essentially anatomical, based on Golgi preparations. Münzner curiously holds that Bethe's fundamental experiment confirms the neurone theory.

Bethe has demonstrated that in *carcinus mænas* (an anthropod) a complicated reflex can take place in an isolated part of the nervous system in which not a single nerve-cell is present. This is absolutely irreconcilable with a theory which says that the nervous system consists exclusively of nerve-cells.

For Semi Meyer's benefit Nissl repeats the real grounds of his rejection of the neurone theory—the granular or fibro-granular ground substance seen in most preparations, the fact that certain clumps of nerve cells are not gray substances and that some

gray substance contains few nerve cells, the fact that a comparison of elective preparations shows the necessity of the postulate of an intercellular substance, the fact that this substance is most developed in the human frontal lobe, the highest nerve-mechanism. Apathy had already demonstrated for invertebrates that the gray substance (= Neuropil) is a compound of nerve-cells, nerve fibres, and of a finely reticular nervous substance (= Elementargitter) which cannot be resolved into cellular elements.

Among those who maintain the neurone theory of Waldeyer, Ramon y Cajal has the greatest authority. Nissl, in one of the most closely reasoned chapters of the book (pp. 124-241), submits Cajal's position to a minute analysis. Cajal's criticism of Nissl centres in his view of the Golgi net (Bethe), which he regards as an intracellular structure, as merely the superficial layer of the cellular spongionplasma. His view of the reticular nature of the nerve-cell protoplasm is derived from double-stained preparations. Nissl, who takes all nerve-cells for his province, shows the defects of such preparations. He insists on the value of his own method, which alone with certainty gives us a microscopical object of study which is in a constant relation to the living cell; this standard preparation of a cell is his cell-equivalent. The best justification of Nissl's method is the fact that a Bethe fibrillar preparation is the negative of a Nissl preparation. In the cell-equivalent the staining parts of the cell body (= Nissl bodies, Tigroids) consist of deeply medium and faintly staining elements. In a Held or Cajal double-stained preparation the medium and faintly staining elements may appear in the tint of the acid stain (or carmine) instead of the base, and their different refractive index may lead to the description of a structure in the non-staining constituent of the cell body which is falsely equated with the non-staining constituent of Nissl's cell-equivalent.

Cajal's description of the cell protoplasm is without correct objective grounds. With his spongionplasma and limiting membrane disappears his interpretation of the Golgi net. In this chapter we have a sketch of the various fixing agents used in nerve histology.

With regard to His, Nissl remarks that the "neurone theory is a theory of the composition of the adult nervous system, but in any case His has not demonstrated the unicellular origin of embryonic axis-cylinders, far less of adult ones.

In the last seven chapters Nissl seeks to estimate the value of the most modern work in so far as it bears on the problem of the relations of nerve cell, fibre and "gray." He discusses Golgi nets, collaterals, neurofibrils, Golgi cells of Category ii.

With regard to neurofibrils, the points which concern us are chiefly the following:—Neurofibrils are never seen to become finer

or split. They end abruptly at the cell-surface over which we cannot trace them (save in the axis-cylinder process). The course of the fibrils shows that dynamic polarisation does not exist. The neurofibrils of the axis-cylinder process pass continuously into the axis-cylinder of a medullated nerve fibre, but the perifibrillar substance of the axis-cylinder process of the cell is not continued into the axostroma of the axis-cylinder. The axostroma is interrupted at each node of Ranvier. The axis-cylinder of a medullated nerve fibre is therefore not a part of a nerve cell. After the loss of the medullary sheath, the axostroma ceases also, and the neurofibrils cannot be traced. Between this spot and the exterior of the Golgi net a specific nervous substance must exist. Golgi preparations and the appearance of parts of apparently non-medullated nerve fibres in the gray have led to the false opinion that after the loss of the medullary sheath the axis-cylinder continues till it reach the neighbourhood of a nerve-cell. Golgi arborisations do not correspond to real structure; they may be due to partial impregnation of the "nervous gray."

As to collaterals: a collateral means a collateral path of neurofibrils. In view of the limited number of the latter, Golgi preparations cannot correspond to real structure. There is no ground to suppose that a collateral can become a medullated nerve fibre. In the brain there are far more fibres than axis-cylinder processes of nerve cells. Collaterals cannot explain the disproportion. It follows that the extracellular development of neurofibrils is a necessary postulate. The two possible sources are the Golgi nets and the "nervous gray." As to Golgi cells of Category ii., their existence is doubtful.

A nerve cell with its dendrites does not directly abut on the "nervous gray," but between cell and "gray" intervenes Bethe's "Golgi net." This is a closely-fitting basket-work, which completely surrounds the cells, leaving only one opening for the exit of the axon. There are many different types of Golgi net. Nissl holds that Bethe's diffuse Golgi net (Cortex, Cornu Ammonis, etc.), is an artefact. On the inner side the cell neurofibrils probably pass into the Golgi net, at the same time undergoing some structural change. On the outer side the relation to the "gray" varies with the type of net; but there is always a structural connection of the net with the "gray." The Golgi nets are not parts of nerve cells, do not belong to the nervous supporting tissue, are not end-nets of axis-cylinders. As an hypothesis, Nissl suggests that the Golgi nets form the medium in which the elements of the "nervous gray" arrange themselves to enter the cell as conducting neurofibrils, while the fibrils which leave the cell similarly resolve themselves in the net into their components. As the neurofibrils of the cells spring from the elements in the Golgi net, so perhaps in part is the origin of

the neurofibrillar tracts of those fibres which have an extra-cellular origin, the rest of such fibres springing from the "nervous gray."

As to the nervous gray, "all that we definitely know of the nervous gray is that it represents a non-cellular specific nervous substance, which possesses arrangements for isolated conduction, and which can carry out nervous work of the most varied kind." As to its anatomical composition, it is at present useless to form a hypothesis. At present the connection between nerve cell, fibre and gray is unknown.

A book which contains answers to individual critics necessarily contains repetitions. With such an intricate subject, repetition is grateful, and "Die Neuronenlehre" is a model of exposition. Cajal can hardly repeat that Nissl's "nervous gray" is a cheap guess, and with its definite establishment Nissl may justly claim to have determined the end of the third period of the neurone theory

C. MACFIE CAMPBELL.

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# Review of Neurology and Psychiatry

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## Original Articles

### **THE RELATIONS OF BULBAR PARALYSIS AND PROGRESSIVE MUSCULAR ATROPHY.**

By JAMES TAYLOR, M.D., F.R.C.P.

It is a matter of common knowledge that cases of degeneration of the lower motor segment or neuron subserving the limbs (cases of progressive muscular atrophy), have frequently associated with the signs of such degeneration, symptoms and signs indicating a similar degeneration in the bulbar nuclei. This is scarcely surprising, for those nuclei are really the lower motor segment or neuron of the structures concerned in articulation and deglutition, and are therefore strictly homologous with the collections of cells in the anterior horns of the spinal cord which innervate the limb muscles. It is much more common to have this association of bulbar paralysis with progressive muscular atrophy in the cases in which there are evidences of degeneration of both the upper and lower motor segment or neuron—cases of atrophy of anterior horn cells and sclerosis of the lateral or pyramidal tracts—so-called amyotrophic lateral sclerosis. As a rule the bulbar paralysis succeeds the muscular atrophy of the limbs, and ultimately leads to death, because these bulbar structures are closely related to processes on which life depends—especially deglutition. But frequently the signs of involvement of the bulbar structures occur without any evidence of affection of the analogous structures in the cord subserving the limbs. It is not improbable that this evidence of limb affection would be forthcoming if the bulbar affection did not so soon end in death. The writer saw some years ago a patient under the care

of Dr Hughlings Jackson who came into hospital unable to articulate and unable to swallow. There was no sign of muscular wasting in the limbs. This patient was kept alive for over two months by feeding with the stomach tube, and before she died there was evident distinct wasting of the thenar and hypothenar and interosseous muscles of the left hand, so that the case was really one of bulbar paralysis and progressive muscular atrophy. An examination of the nervous system revealed marked degeneration not only of the cells of the bulbar nuclei but also of those in the cervical enlargement, and to a much less marked degree in the lumbar enlargement of the spinal cord. Had this patient not been fed artificially and so kept alive she would have been regarded clinically, at least, as an example of simple bulbar paralysis.

The following case of a man whom I first saw as an out-patient at Queen Square illustrates the same association. In his case, however, the bulbar affection was slow in its course, and was not so severe as to necessitate the use of the stomach tube, yet, as the bulbar symptoms were the early ones, he has now, without anything like complete paralysis of articulation and deglutition, very distinct atrophy of the small muscles of the hands. He was admitted as an in-patient under the care of Dr Hughlings Jackson, who has kindly permitted me to describe the case. Indeed I owe it to Dr Jackson that my attention was first directed to this subject during the treatment of the other patient to whom I have referred, who was under his care when I was his house physician.

The patient now under notice was a man of 64, without anything very significant in his previous health except syphilis twenty-five years ago and ague in Calcutta at one time. He was hard-working, sober, industrious, and in excellent health until twelve months before his admission. He then began to have difficulty in articulating and a few months later in swallowing. He soon afterwards noticed a clumsiness and difficulty in using his hands. His speech became increasingly difficult and indistinct and he had frequent choking attacks when swallowing. On admission he presented a very much wasted tongue, wasting of the small muscles in each hand, characteristic bulbar articulation and the emotional instability so frequently present when bulbar structures are affected. He also had very active knee-

jerks, wrist-jerks, and jaw-jerk, and he had ankle clonus, so that the upper motor segment was evidently also degenerating. He improved a good deal in hospital under the influence of hypodermic injections of strychnine.

The case may seem to some comparatively unimportant. I venture to think, however, that it is not without interest as tending to widen our conceptions of disease and as illustrating the essential identity of the morbid processes in several different diseases—bulbar paralysis, progressive muscular atrophy and amyotrophic lateral sclerosis.

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### ON AN UNUSUAL CAUSE OF FACIAL PARALYSIS.

By LEONARD WILLIAMS, M.D., M.R.C.P.,  
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THE etiology of the majority of cases of facial paralysis is admittedly obscure. The label "rheumatic" has been affixed to these cases, not because anyone seriously believes that they have any relation to acute rheumatism or its congeners, but because they are said to be due to "chill," with which rheumatism is, in some confused way, supposed to be related. It is not my present purpose to endeavour to drive any more nails into that coffin which the advance of knowledge is preparing for the "chill" theory, but I may be permitted to point out that the occurrence of the phenomena of "chill"—that is, shivering and depression—when they mean anything at all, signify that the disease which is subsequently to be ascribed to the "chill" has already successfully invaded the patient; that the phenomena proclaim the success of the invasion as surely as the rash or the evidences of inflammatory action which develop later. My present purpose is rather to endeavour to show that in spite of the fact that the chill theory is untenable, there may nevertheless be some association between the rheumatic diathesis and the causation of facial paralysis.

This association, if, indeed, it can be said to exist at all, is to be traced through the analogy of another disease of the nervous system, namely chorea. The connection between the latter and true rheumatism now admits of no doubt. It has been proved clinically over and over again, and comparatively recently Poy-

ton and Paine\* have clinched the matter by showing their bacteriological identity. In a large number of cases of chorea, however, the direct determining cause is fright or some other emotion, and in these it is necessary to assume that rheumatism plays the part of an antecedent and latent cause, except for which the element of emotion would be inoperative. It is quite certain that all frights, even in neurotic children, do not result in chorea.

I recently came across a case which suggests that a similar explanation might account for some at least of the cases of facial palsy which are due to a neuritis within the Fallopian tube. This was that of a healthy-looking young woman of twenty-five years of age, who presented herself at the hospital one Monday afternoon, with a left-sided facial paralysis, and the following story:—She had always enjoyed robust health, and no member of her family had ever, to her knowledge, suffered from articular or other forms of rheumatism. On the foregoing Saturday her six months old baby had slipped from her arms and fallen downstairs. She was very much alarmed at the time, but as the child was, happily, uninjured, she forgot all about it until the next morning, when her husband remarked that "her face was all crooked." Although pressed closely upon the point, she would not admit that there had been any exposure to cold or chill, and was quite satisfied that the fright she had received was the sole cause of her condition. The symptoms presented nothing worthy of remark. They were those of a typical Bell's palsy, slight in degree, which in the course of a fortnight disappeared completely.

Apart from any theories as to etiology, this case seems worthy of recording, because, so far as I have been able to ascertain, not only is emotion not recognised in the text-books as a cause of the complaint, but no similar case is to be found in the literature of the subject.

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#### **NOTE ON LATERAL TONGUE-MOVEMENTS.**

By LEONARD J. KIDD, M.D. Brux., &c.

MORE than a year ago I discovered accidentally on myself, and have since then verified the observation on others, that in the

\* *Lancet*, May 4, 1901.

performance of lateral tongue-movements at and below the horizontal plane of the tongue it is easy to both see and feel a contraction of the infra-hyoid muscles of the same side, viz., the sterno-hyoid, sterno-thyroid, and omo-hyoid (especially the posterior belly).

The point was not mentioned by any of the authorities I consulted.

When, soon afterwards, Professor Cunningham's "Text Book of Anatomy" appeared, I found on p. 388 that Professor A. M. Paterson leads one to infer that the infra-hyoid muscles, when acting on one side only, help in lateral movements of depression to that side.

In the study of these movements the following points must be attended to :—

- (1) Put the patient in a good light, with neck quite exposed to view.
- (2) Stand three or four feet from him; in self-observation half that distance from a well-lighted mirror.
- (3) Avoid excessively powerful lateral movement, for this will bring out the action of the platysma muscle, and will spoil the test.

The posterior belly of the omo-hyoid can be by this method mapped out; a knowledge of this may prove of help to operating surgeons.

Lesions involving the nerves that supply these muscles (first and second cervical) or their roots are rare. Still I think the points I have mentioned are worthy of clinical study.

The editor of this Review has kindly drawn my attention to Dr James Collier's article in the August number, in which, on p. 530, he mentions that on forward bending of the head against resistance the posterior belly of the omo-hyoid becomes prominent.

## Abstracts

### ANATOMY.

**ON THE SO CALLED "GYRUS HIPPOCAMPI"** G. ELLIOT SMITH,  
(306) *Journ. Anat. and Phys.*, vol. xxxvii., 1903, pp. 324.

THE object of this note is to emphasise the fact that the so-called hippocampal gyrus is composed of three kinds of pallium—lobus pyriformis, hippocampus and neopallium—which present the most fundamental differences in their structure, in their developmental history and probably in their functions. It is proposed, therefore, to separate these parts, which have so little in common, and discard the terms "gyrus hippocampi" and "gyrus uncinatus." The anterior part, representing the pyriform lobe of other mammals, may be distinguished as the "area pyriformis." It is part of the olfactory area and is specially modified in structure to act as a receptive organ for the olfactory impulses, which are conveyed to it by an uninterrupted path composed of the fibres of the lateral olfactory tract (springing from the "mitral cells" in the olfactory bulb). The rest of the so-called hippocampal gyrus is named "gyrus paradentatus" and differs in a most fundamental manner from the lobus pyriformis and hippocampus. It is a part of the general cortex or neopallium.

The reason for the confusion of the pyriform and neopallial parts in this region of the human brain is due to the fact that in man the rhinal fissure (which marks the extent of the pyriform area) is subject to much irregularity and is commonly regarded as part of the collateral sulcus (*vide* Cunningham's "Text-Book of Anatomy," p. 518). The rhinal fissure includes not only the incisura temporalis, but the anterior part of the so-called collateral sulcus (in Cunningham's figure 386, p. 518, so much of the sulcus as lies alongside the word "collateral").

AUTHOR'S ABSTRACT.

**RESEARCHES ON THE GRANULES AND COLOURABLE COR-  
(307) PUSCLES OF THE CELLS OF THE CENTRAL AND PERI-  
PHERAL NERVOUS SYSTEM.** G. MARINESCO, *Zeitschr. f.  
allg. Physiol.*, Bd. iii. H. 1, 1903.

IN this paper, Marinesco describes an extensive series of histological observations upon the acidophile granules of the protoplasm of the nerve cells, and compares his results with those of previous writers on the subject. He adds a description of a special feature observed in the nucleus of certain nerve cells.





The material studied was obtained from man and the dog, and was fixed in various ways. The sections were stained with acid and basic dyes and by the special methods of Ehrlich, Biondi, Romanowski, and Benda. Preparations from many different situations showed, in the protoplasm of the nerve cells, granules varying in number, form and volume according to the age of the subject, but having the common character of staining with acid dyes, used alone or in the mixtures of Ehrlich and Biondi. These granules were most abundant in the cells of the lumbar, sacral and cervical ganglia, less numerous in those of the dorsal ganglia, and still less so in those of the brain. They were also found in the cells of the sympathetic ganglia, *locus caeruleus*, and some other situations. They were not to be seen in all of the cells, but were constantly present in the large cells. In the cells of the spinal ganglia of a child of two years, they appeared as fine, discrete granules, varying somewhat in size, and of a violet red tint in preparations by Romanowski's method. They formed a more or less dense mass, often situated in the neighbourhood of the nucleus. The mass was most commonly single, but not infrequently there were two or even three in one cell. At the age of five years the granules formed a denser mass, they were more numerous and especially more voluminous and unequal. The mass was situated sometimes in the neighbourhood of the nucleus, sometimes near the periphery of the cell. In some instances the granules were lying in an amorphous substance of a yellowish colour. In certain cells the acidophile granules were mixed with black pigment granules. In the adult they appeared as granules or corpuscles of unequal size, disseminated throughout the body of the cell, or collected into one or two, more or less dense, masses. In the cells of the sympathetic ganglia, the granules presented essentially the same appearances. In view of certain conclusions of Olmer, the author has made a special study of these granules in the nerve cells of the *locus caeruleus*, and has found that they have essentially the same characters and staining reactions as in other situations. He therefore dissents from the conclusion of Olmer that these nerve cells contain amphophile granules that are essentially different from the erythrophile granules which he (Marinesco) described in 1899. He maintains that these erythrophile, acidophile or oxyneutrophile granules are also to be identified with the bioblasts of Altmann, the fuchsinophile granules of Levi, and the neurosomes of Held. He makes, however, some reservations in regard to their correspondence with the last named.

Discussing the question of the physiological significance of these granules, the author suggests that they are of the nature of diastase and concerned with the vital processes that take place in the nerve cells.

The special feature that he has observed in the nuclei of certain nerve cells consists in the presence of bodies which he has designated *corpuscles acidophiles paranucléolaires*. He has found them almost exclusively in the cells of the *locus niger* and *locus caeruleus*. In preparations by Romanowski's method, they are coloured in various shades of red. When stained with certain acid dyes, such as acid fuchsine, they take a tint appreciably distinct from that assumed by the nucleolus. They do not give the reactions of fatty matter. As a rule, they have a homogeneous aspect. Their number varies from one to six. Their position in relation to the nucleolus is not constant. In size they are generally rather smaller than the nucleolus. The author remarks that these corpuscles are found in nerve cells that contain melanin, and suggests that they are connected with the formation of this pigment.

W. FORD ROBERTSON.

**REMARKS ON ARTEFACTS WHICH STAIN BLACK IN MARCHI  
(308) PREPARATIONS.** E. STRANSKY, *Neurolog. Centralbl.*, July  
15, 1903.

STRANSKY draws attention to the presence of Marchi-staining particles in the fibres of peripheral nerves which have been injured in their removal, or in the proximity of even careful section. These granules are not only found in the retracted part of the fibre whose calibre is irregular, but spiral and comma-shaped masses, as a rule parallel to the long axis of the fibre and never extending across its whole diameter, may be visible where the fibre is otherwise apparently normal.

The importance of recognising these as artefacts in experimental work, and especially when the effects of toxins on the nervous system are to be investigated, is evident, but it is easy when attention is paid to their shape and character.

GORDON HOLMES.

**PHYSIOLOGY.**

**MUSCULAR MOVEMENTS AND THEIR REPRESENTATION IN  
(309) THE CENTRAL NERVOUS SYSTEM.** *The Croonian Lectures.*  
By CHARLES E. BEEVOR, M.D., F.R.C.P.Lond.

LECTURE I.

IN introducing his subject, Dr Beevor gives his reasons for preferring the physiological to the anatomical or electrical methods of investigating the part played by individual muscles in the

execution of voluntary movements. He points out that the latter methods only teach us what a muscle *may* do, and that they throw no light on what a muscle *does* do when brought into action under the influence of the will.

Pursuing the physiological method the lecturer studied, both in normal persons and in patients suffering from various forms of paralysis, the muscular contractions associated with: (1) the movement of an unburdened limb, (2) the movement of a limb loaded with different weights, and (3) the attempt to move a limb fixed by an opposing force. In these procedures the amount of work done was measured by spring balances or by the traction scale of a clinical dynamometer.

While many of the results of this investigation are in harmony with those of other observers, in a number of instances Dr Beevor comes to different conclusions and, wherever this is the case, gives ample and strong reasons in support of his views. For the details of this reasoning reference must be made to the original lectures, and only the conclusions arrived at can be summarised here.

*Flexion and extension of the fingers.*—The first phalanges of the fingers are flexed on the metacarpal bones by the interossei and lumbricales and also by the flexores digitorum on their way to the terminal digits. If, however, it is required to flex the last two phalanges of each digit, but not the first, the extensor communis digitorum comes into play—a muscle which extends the first more than the last two phalanges. If flexion of the fingers is required without any movement of the wrist, then the extensors of the wrist also contract.

Extension of the last two phalanges is produced mainly by the interossei and lumbricales, but in their absence, and during passive flexion of the first phalanges, the movement can be executed by the extensor communis digitorum. Extension of the proximal phalanges is accompanied by contraction of the flexors of the wrist.

*Lateral movements of the fingers.*—Adduction is performed by the palmar interossei, and abduction of the three large fingers by the dorsal interossei. Abduction of the index finger alone necessitates some action on the part of the extensores ossis metacarpi and primi internodii pollicis in order to prevent movement of the latter digit.

Abduction of the little finger is performed by the special abductor minimi digiti, but, as this arises from the pisiform bone, it is accompanied by some contraction on the part of both the flexor carpi ulnaris and extensor ossis metacarpi pollicis, the former steadying the pisiform bone and the latter counteracting the consequent tendency towards flexion of the wrist.

*Movements of the thumb.*—Flexion of the terminal phalanx is performed by the flexor longus pollicis, and, when it is the only

movement required, the proximal phalanx is fixed by the extensor primi internodii. Extension of the terminal phalanx necessitates action on the part of not merely the extensor secundi internodii but also of the abductor, adductor and flexor brevis pollicis, owing to the tendinous connections of the latter muscles with the former.

Flexion of the metacarpal bone is caused by the opponens, abductor, adductores and flexor brevis pollicis when flexion of the first phalanx is also required, but when flexion of the metacarpal bone is accompanied by extension of the phalanges the extensor primi internodii is all brought into action.

Extension of the metacarpal bone is performed by the extensor ossis metacarpi and also by the extensor primi internodii, and when this movement is accompanied by extension of the first phalanges both the flexor and extensor carpi ulnares are made to contract.

In abduction of the thumb the abductor pollicis, the opponens pollicis, the outer head of the flexor brevis pollicis, the extensores ossis metacarpi and primi internodii are all brought into use. Although it is possible to abduct the thumb when its extensors are paralysed, it is quite impossible for the latter muscles to perform the same movement in the absence of help from the small muscles of the thenar eminence.

Adduction of the thumb from the position of extreme abduction is carried out by the adductores and extensor secundi internodii pollicis, but in the course of the movement these muscles are joined by the flexor carpi ulnaris with the probable object of counteracting the tendency to extension of the wrist. Opposition is best performed after abducting the thumb, and the muscles which take part in this movement are the abductor, opponens, adductores and flexor brevis pollicis.

*Combined movements of fingers and thumb.*—In grasping an object the flexors of all the different digital joints are brought into action, and along with them the extensors of the wrist are made to contract with a force which is proportional to the energy of the grasp.

*Movements of the wrist.*—Flexion is carried out by the flexor carpi radialis, the flexor carpi ulnaris, the palmaris longus, and by the flexores digitorum when resistance is opposed to the phalanges. The extensor ossis metacarpi pollicis is also a flexor of the wrist. Abduction of the wrist is brought about by the action of the flexor carpi radialis, the three extensors of the thumb, and by the extensor carpi radialis longior; adduction by the flexor and extensor carpi ulnares. When the fingers are flexed so as to form a fist, extension at the wrist is obtained by contraction of the three proper wrist extensors, and to a slight degree by the help of the extensor secundi internodii pollicis. If, however, the fingers are fully extended in

the first place, then extension of the wrist is accomplished by the extensor communis digitorum, and the latter muscle is not assisted by the extensors of the wrist proper until a resistance of some three or four pounds has to be overcome. In this way the inability to extend the wrist, except when the fingers are flexed, in cases of paralysis of the extensors of the fingers, due to plumbism, is shown to be of physiological origin. An analogous condition obtains when flexion of the carpus is performed by the flexores digitorum, the fingers being already flexed. In this case, too, the proper flexors of the wrist do not come to the assistance of the flexors of the fingers until a considerable opposition is afforded to the movement. With regard to the synergic contraction of the flexors of the wrist with the extensors of the fingers and of the extensors of the wrist with the flexors of the fingers, the following explanation is given by Dr Beevor. When a muscle by passing over two or more joints has two or more different actions, then, if only one of these actions is required, other muscles are brought into the movement whose actions are antagonistic to those movements of the muscle which are not required. This rule applies, as far as the lecturer has been able to observe, throughout the muscular system.

#### LECTURE II.

*Movements of supination and pronation at the radio-ulnar articulations.*—The muscles producing pronation are the pronator teres and the pronator quadratus, and, if resistance to the movement be applied to the hand, the flexor carpi radialis and palmaris longus also contract.

Supination is performed by the supinator brevis, the biceps, and, when resistance is applied to the hand, by the extensores carpi radiales longior and brevior and by the extensors of the thumb. The supinator longus or brachio-radialis contracts slightly in very strong pronation, but otherwise is not concerned in the movements at the radio-ulnar joints. In opposition to Duchenne, Dr Beevor points out that the biceps can take part in supination without flexing the elbow, and that this is possible owing to the synergic action of the triceps, especially of the inner and outer heads of that muscle. When both supination and flexion are required the triceps ceases to contract, and when the triceps is paralysed supination is not possible without some degree of flexion at the elbow. Since the pronator radii teres is also a slight flexor of the elbow, pronation of the forearm is accompanied by slight contraction of the triceps.

*Movements of flexion and extension at the elbow joint.*—The muscles taking part in flexion are the biceps, the brachialis anticus, the supinator longus, the pronator radii teres, and, when

the hand is closed, there is very slight action on the part of the flexors of the wrist.

Extension of the forearm on the arm is carried out by the triceps and anconeus unaided by any action on the part of the extensors of the wrist.

*Movements of the humerus and scapula.*—In advancing the humerus the anterior fibres of the deltoid, the clavicular fibres of the pectoralis major, the biceps and probably the coraco-brachialis contract and carry the humerus forward nearly to the horizontal line. This action tends to rotate the scapula with the acromion downwards and to push its inferior angle towards the spinal column, but this is prevented by the contraction of the acromial and—Dr Beevor considers—the inferior fibres of the trapezius. The deltoid and other muscles cannot carry the humerus further than the horizontal line; after passing through 45 degrees the serratus magnus comes to their assistance, and, drawing the lower end of the scapula forwards, raises the acromion with the humerus so that the arm is elevated to the vertical position. When this action is attempted with a paralysed deltoid or with a fixed shoulder joint the serratus acts at once and the scapula moves outwards at once, so much so that this movement of the scapula is a diagnostic sign of one of these two conditions.

Winging of the scapula may be caused by paralysis of the trapezius, especially of its lower fibres, as well as by paralysis of the serratus magnus. When due to paralysis of the former muscle the deformity is slight, reaches its maximum on the humerus being advanced through 45 degrees and then disappears, while the arm can be elevated to the vertical position. With paralysis of the serratus magnus the winging increases as the arm is advanced to the horizontal position, beyond which it cannot be raised.

The middle fibres of the deltoid and the supraspinatus abduct the humerus, and the same conditions are met with in regard to the movements of the scapula as in advancing the arm.

Depression of the humerus in the antero-posterior plane from the vertical position above the head through 180 degrees is performed by the sternal fibres of the pectoralis major, the pectoralis minor, the latissimus dorsi, the teres major, the teres minor, the infraspinatus, the long head of the triceps and perhaps by the subscapularis. This action of the long head of the triceps is very well seen in cases where the triceps is unaffected while the flexors of the elbow are paralysed. Every time the patient depresses or adducts the humerus the elbow is extended by the unopposed long head of the triceps. Retraction of the hanging humerus posteriorly to the vertical line is performed by the latissimus dorsi, the teres muscles and by the posterior half of the

deltoid. The pectoralis minor also takes part, but not the pectoralis major, so that the movement is a means of separating the actions of these two muscles. Adduction of the humerus is carried out by the same muscles as take part in depressing the arm with the addition of the clavicular fibres of the pectoralis major and the posterior fibres of the deltoid.

In both depressing and adducting the humerus the scapula has to be fixed by the rhomboids and the lowest fibres of the trapezius, so that when the former are absent the inferior angle of the scapula is drawn into the axilla by the teres muscles, and when the lowest fibres of the trapezius are paralysed the scapula is lifted up by the rhomboids and teres major and minor. In depression and in adduction of the humerus from the horizontal line there is contraction of the abdominal muscles, especially of the rectus abdominis and obliquus externus, which fix the ribs and prevent them being drawn up by the pectoralis major. In depressing the arm both recti act, but the homolateral one more than the other; in adducting the arm only the homolateral rectus takes part, but there is also some contraction on the part of the erector spinæ of the same side.

Horizontal adduction of the arm toward the median line of the body is carried out by the anterior fibres of the deltoid, the coracobrachialis and by the pectoralis major.

In horizontal abduction of the arm the different fibres of the middle part of the deltoid successively come into action from before backwards until the posterior fibres act together with the latissimus dorsi, subscapularis and teres muscles. Associated with this is the fixation of the scapula by the trapezius, in which all but the clavicular and acromial fibres take part. This is the best movement to bring out the middle fibres of the latter muscle.

The internal rotators of the humerus are the pectoralis major, the anterior fibres of the deltoid, the teres major, the latissimus dorsi and probably the subscapularis.

The external rotators of the humerus are the teres minor, the infraspinatus and posterior fibres of the deltoid.

During internal rotation with the humerus horizontal the rhomboids fix the scapula; during external rotation the lowest fibres of the trapezius perform this function. Consequently in paralysis of the trapezius there is great displacement of the scapula with external but not with internal rotation.

*Movements of the scapula independent of the humerus.*—Elevation is performed by the trapezius, especially by its acromial fibres, and by the levator anguli scapulæ. The levator anguli scapulæ sometimes takes part with the serratus magnus in advancing the scapula and perhaps it counteracts the rotation of the latter, with the acromion upwards, by the serratus magnus.

*Movements of the spinal column.*—The flexors of the spine are the recti abdominis with the pyramidales and external obliques. The complete movement of sitting up from the recumbent position consists of two stages. In the first the sternum is approximated to the pubes by the recti abdominis flexing the lumbar spine; in the second the pelvis with the spine is flexed on the femora by the psoas and iliacus and other flexor muscles. During the movement of sitting up in health the umbilicus does not alter its position; but when the lower parts of the recti are paralysed from a lesion of the spinal cord, or its nerves, below the level of the tenth dorsal segment, the umbilicus is raised by the upper parts of the recti, sometimes to the extent of an inch. In two cases Dr Beevor has seen the umbilicus drawn downwards in the same movement owing to weakness of the upper parts of the recti.

The extensors of the spine are the erectores spinæ and their subdivisions.

*Movements which regulate and maintain the erect posture.*—In the erect position there is very little contraction to be felt in the erectores spinæ or in the recti abdominis, but when the centre of gravity is displaced by a forward movement of the trunk the erectores spinæ instantly contract and the recti abdominis relax. In the same way on extending the head backwards the recti abdominis contract and the erectores spinæ relax.

But if it is desired to bend the trunk gradually forward, the contracted erectores slowly relax and let out the weight of the trunk in the same way as a heavy weight is slowly lowered to the ground by a crane.

In lateral flexion to one side where resistance has to be overcome, the erector spinæ and rectus abdominis of the same side can be felt to contract together with the external oblique and probably the quadratus lumborum; but in inclining the trunk to one side with no resistance to be overcome, the muscles of that side are relaxed and the muscles of the opposite side—the antagonists—contract.

In rotation of the spine to the right the left external oblique is the only muscle which can be felt to come into action.

In conclusion, Dr Beevor gives his reasons for thinking that the contraction of the erectores spinæ which takes place when the arm is advanced to the horizontal position by a person in the erect position is not an essential part of the movement.

E. FARQUHAR BUZZARD.



**PATHOLOGY.**

**HISTOLOGY OF GENERAL PARALYSIS.** *Congrès des aliénistes et (310) neurologistes de France et des pays de langue française.* Bruxelles, août 1903. Rapport présenté par le Dr KLIPPEL.

In this monograph of 128 pages the author describes what he regards as the essential histological lesions of general paralysis, and discusses various questions in the pathogenesis of the disease. He attaches special importance to the well-known cellular infiltration of the walls of the cerebral vessels, which he describes as a diapedesis of white blood corpuscles, and makes virtually the basis of a classification of the different forms of general paralysis. He divides the disease into: (1) inflammatory general paralysis, characterised by diapedesis of leucocytes into the walls of the cerebral vessels; (2) associated general paralysis, in which this inflammatory encephalitis is grafted on to pre-existing cerebral lesions, such as those produced by chronic alcoholism, atheroma, syphilitic gummata, etc.; and (3) degenerative general paralysis, in which the vascular and nerve cell lesions are purely degenerative and diapedesis is absent.

The work is divided into two parts, the first of which deals with the histological lesions, whilst the second is devoted to a statement of the author's conclusions. The first part is composed of six chapters. In the first of these the author gives an account of the morbid changes found in inflammatory general paralysis, briefly describing the lesions affecting the skull, membranes and cerebral substance. He holds that erosions are dependent upon the presence of a small focus of softening, the result of obliteration of a vessel affected by the inflammatory process. He maintains that diapedesis and not sclerosis is the characteristic change in the walls of the vessels. The cells that have passed out from the lumen of the vessel consist of lymphocytes and polynuclear leucocytes. This diapedesis of white corpuscles has not, however, a specific character; it is met with in bacterial inflammations of other organs. Following a description of the changes affecting the cortical nerve cells, neuroglia, basal ganglia, etc., an account is given of the condition of the cerebro-spinal fluid. Some post-mortem bacteriological investigations are here recorded in which the author isolated from this fluid various pathogenic bacteria, including the pneumococcus, staphylococci, streptococci and the tubercle bacillus. In many cases the results of the bacteriological examination were negative.

In the next two chapters, associated general paralysis and degenerative general paralysis are described and their nature interpreted in the manner already indicated. The fourth chapter

deals with the lesions of the spinal cord and nerves. The broad conclusion arrived at is that the spinal lesions have a two-fold origin, being either primary localisations of the disease in the cord or degenerations secondary to the encephalitis.

The next chapter treats briefly of the lesions of the viscera and sympathetic. The author gives a summary of an unpublished paper which records the results of an investigation of the state of the viscera in 36 autopsies. On the ground of these researches he distinguishes four varieties of lesions, namely: (1) those that have existed prior to the onset of the general paralysis, such as sclerosis of the liver, interstitial nephritis, etc.; (2) those dependent upon lesions of the nervous system and manifesting themselves in capillary ectases and hæmorrhages from the kidney, liver and lung; (3) those connected with the state of marasmus, more particularly fatty degeneration and passive congestion of the organs; and (4) those dependent upon secondary infections by pneumococci, streptococci, staphylococci, etc., and consisting mainly of pneumonias, nephritis, and eschars. He emphasises the importance of lesions of the second group, namely, the vaso-paralyses, because of their more direct dependence upon the lesions of the nervous system. After describing the changes of this nature in the lungs, heart, liver and kidneys, he concludes that in all the viscera the process is the same, being marked by capillary congestion, miliary hæmorrhages and pigmentary degeneration, atrophy and degenerative catarrh of the neighbouring epithelium. The gastro-intestinal tract is not mentioned.

The sixth chapter deals with the histological alterations of the blood, which the author has studied in conjunction with Lefas. The following are some of the chief results recorded. The existence of a true leucocytosis could not be established at any stage of the disease. In the first period, the red corpuscles are normal in number, there is an increase in the percentage of neutrophile polynuclear leucocytes and diminution in that of the mononucleated leucocytes. There are abnormal cell-elements (one to three per cent.), which are probably either macrophages or degenerated leucocytes. There is no eosinophilia. Nucleated red corpuscles are common. In the second period, the most important further change consists in diminution in the percentage of polynuclear leucocytes and an increase in that of the mononucleated leucocytes. In the third period the conditions do not differ materially from those of the second.

In the second part, the author states his conclusions regarding respectively the nature, consequences and development of the lesions described. General paralysis, he says, is a *syndrome*. A *syndrome*, however, does not correspond either to a particular pathogenic cause or to a single lesion, but to a special localisation

and to similar reaction to various causes and lesions. Hence cases clinically similar may have a different pathology, in accordance with his classification of the disease into three groups. The author does not favour the theory of the essentially syphilitic origin of general paralysis. He asks the supporters of this theory, What is the nature of this disease which they call parasymphilitic? If they mean that it is an infection favoured by a previous syphilis, he says he is in agreement with them in regard to a considerable number of cases. The ground upon which he essentially bases his contention that his inflammatory form of the disease is the result of an active infective process is that of the resemblance of the histological lesions to those that we know to be produced by bacteria. He appears to regard the infection as intracranial, but like others who have hazarded a similar opinion, he has made no observations that serve to prove it.

In considering the consequences of the cerebral lesions, the author distinguishes those that are of an irritative character and therefore manifest themselves by perversion of the functions of the nervous elements, and those that are destructive, implicating the functional connections between the different elements and therefore entailing especially the condition of dementia. Coming, lastly, to the development of the lesions, he discusses the time-worn question of which tissue of the brain is primarily affected, and concludes that the morbid process starts both in the vessels and nerve cells.

W. FORD ROBERTSON.

**CHROMATOLYSIS IN THE CELLS OF THE ANTERIOR CORNUA (311) OF THE SPINAL CORD.** KARL BRAEUNIG, *Arch f. Anat. u. Physiol.*, H. 3 u. 4, p. 251.

THE writer, after giving an account of the various theories which have been advanced to explain the degeneration of motor cells in the spinal cord when the corresponding peripheral nerve has been cut or seriously injured, records some experiments which he has conducted to determine whether the changes can be attributed either to the diminution of impulses descending to the cell through the upper neuron, or of those reaching it reflexly from the sensory fibres of the injured limb.

He has performed one experiment to elucidate the former possibility, and reports that in a dog killed sixteen days after destruction of the motor cortex cerebri no changes were observed in the anterior cornual cells. A series of experiments in which certain of the lumbar sensory roots were divided show that both in the frog and the dog degenerative changes subsequently occur. These were most marked on the side on which the sensory root

had been divided, but were also detected in smaller numbers on the opposite side. The cells most affected in the dog were those of the lateral groups, though in advanced cases altered cells appeared in parts of the anterior and central groups also. The changes were most conspicuous in those segments whose cells received sensory reflexes from parts of the body such as the foot and toes, where the influence of stimuli may be supposed to be greatest and most important, and therefore his observations support the view expressed by Warrington, although the degenerating cells were not so strictly limited to these segments in his experiments as in those recorded by the latter observer. The author gives a fairly complete record of the German literature dealing with this subject.

HARRY RAINY.

### PSYCHOLOGY.

#### NOTES ON THE PSYCHOLOGY OF BACKWARD CHILDREN.

(312) TOBIE JONCKBEERE, *Arch. de Psychol.*, June 1903.

M. JONCKBEERE remarks that the training of backward and mentally feeble children should be based upon an understanding of their psychology. In this article he sums up the observations which he has made during a number of years upon this subject.

*Sensation.*—The organs of sense are frequently defective in backward children. This necessarily interferes with the proper development of the association centres, and influences the whole mental life of the child.

*Muscular Sense.*—The author's experience confirms Féré's views as to the influence of training in voluntary movement upon sensibility and upon mental activity in general. Backward and feeble-minded children are defective in muscular sense, and accordingly, such manual training as will tend to correct this fault should form an important part of their education.

*The Illusion of Weight.*—Of two objects of equal weight, but of different sizes, the smaller feels the heavier. This illusion of weight is frequently present in feeble-minded children as it is in normal persons. Its absence is of grave significance, indicating that the child belongs to the pathological type (*arriéré médical*).

*Movement.*—Muscular movement plays a very important rôle not only in the development of the brain but in mental evolution. According to Féré the energy and precision of the movements are in direct relationship with the vividness of the mental representation of these movements and with the degree of mental development. In feeble-minded children, therefore, gymnastics are absolutely essential, but the difficulty of teaching gymnastics is



very great on account of the lack of attention and the feebleness of the will. The way to get over this difficulty is musical drill. Through the influence of music the disorderly movements of the children become regular and co-ordinate.

*Colour Sense.*—Colour sense is usually defective, but in *arriérés pédagogiques* it can often be cultivated without much difficulty, while in *arriérés médicaux* it is impossible, or at any rate extremely difficult, to cultivate it to even a small degree.

*Lying.*—Mentally feeble children are notoriously untruthful, and the author gives a number of examples to show how careful one must be in giving credence to their assertions.

*Special Cases.*—A few notes are added upon special cases ; upon the visual memory of a feeble-minded child ; upon a case of mental enfeeblement following meningitis in which, although there was practical arrest of mental development, two languages were acquired in addition to the German originally spoken ; upon cases in which arithmetic was readily acquired by children who made little progress with reading, and others in which reading was learned with comparative ease while arithmetic remained in abeyance. The idea of space is said to be acquired more easily than that of time, and it is stated also that the feeble-minded understand the idea of "to-morrow" much more readily than that of "yesterday."

W. B. DRUMMOND.

**THE MEASUREMENT OF ATTENTION IN FEEBLE-MINDED  
(313) CHILDREN.** F. CONSONI, *Arch. de Psychol.*, No. 7, June 1903.

THE author undertook this research with the objects of comparing the power of attention of feeble-minded with that of normal children, and of ascertaining to what extent one could apply to them the æsthesiometric method which Griesbach made use of in studying mental fatigue in school-children. It seemed possible that such a research might be of practical as well as scientific interest, as a step towards obtaining a method of gauging the degree of mental enfeeblement, and perhaps also the degree of educability of the feeble-minded.

The æsthesiometric method of gauging attention rests upon the principle that the results of æsthesiometry, at one time regarded as an exact measure of the peripheral factor in sensation, are in reality so directly related to the state of attention that they may be made use of as a measurement of the latter.

In his observations the author made use of the æsthesiometer of Griesbach (a description of which is to be found in *Pflüger's Archives*, June 1897), the special advantage of which is that the least degree of pressure upon the skin can be regulated.

The observations were made upon 15 children, 11 of whom were feeble-minded, while the rest were normal. Preliminary experiments were made daily at the same hour for 20 days in order that the subjects might get used to the method and understand what was required of them. Thereafter a fresh series of experiments was begun for the purpose of investigating both static (fixed) and dynamic (distributive) attention. The latter was studied by requiring the subject to count the touches of a hammer upon his left hand, or the beats of a metronome, while the investigator experimented with the usual technique upon the right index finger.

The conclusions reached by the writer are: That his method gives excellent results in the study of discriminative tactile attention. That a certain amount of conative tactile attention is always possible except in very severe cases. That in feeble-minded children such attention is always defective in some of its qualities (rapidity, constancy, etc.) or in all. That such alterations are more marked when the feeble-mindedness is of a high degree. That dynamic attention may attain a sufficient degree of rapidity, but is constantly lacking in extent. That the degree of general capacity for attention is related to the emotional temperament and the powers of inhibition. That there is also a direct relationship between this capacity for attention and the degree of feeble-mindedness—that is to say, that an examination of the faculty of attention affords a valuable index to the mental state, and is in itself almost sufficient for a diagnosis of the degree of mental enfeeblement.

W. B. DRUMMOND.

### CLINICAL NEUROLOGY.

**TABES DORSALIS: A STUDY OF 140 CASES OF LOCOMOTOR**  
(314) **ATAXIA.** JOSEPH COLLINS, *Med. News*, January 3, March  
7-14, June 13-20, 1903.

IN the first paper, after an interesting historical account of the recognition and interpretation of the disease, Collins deals with the *etiology* of tabes. Of his 140 cases, 124 were men, 16 women, *i.e.* a ratio of about 7.5 men to 1 woman. The average age of onset was 38½ years—the youngest 18, the oldest 60; 50 per cent. of the cases among women occurred between the ages of 20 and 30, while only 11 per cent. of the men were in this decade. Of the 124 male patients, 85 (*i.e.* 68 per cent.) gave a definite history of syphilis; in 19 others a luetic infection seemed probable, and 12 others had gonorrhœa—therefore, 116 of the 124 cases (94 per cent.) gave very suggestive evidence of, or admitted having had some form of venereal disease. Of the 16 female patients, 11 (*i.e.* 70 per



cent.) had positively had syphilis, and some of the others had suspicious signs. Collins strongly advocates the view that syphilis is the cause of tabes, basing his contention on these facts: (1) the great frequency of a history or evidences of syphilis in cases of tabes—he found that a specific history occurred ten times oftener in his series of tabetic cases than in a corresponding number of cases of other nervous diseases; (2) the occurrence of conjugal tabes; (3) cases of so-called hereditary tabes—he believes that these cases are due, not to direct transmission of the disease, but to syphilis inherited from the parent; he cannot find on record a single genuine case of infantile tabes in which syphilis did not exist. He does not think that the other supposed ætiological factors play an important rôle in the causation of tabes, *e.g.* sexual excesses, exposure to cold and wet, alcoholic excess (present in 42 of his 140 cases), “the strenuous life,” trauma. In only one of his cases was there any real ground for considering it of traumatic origin, and probably the injury only hastened the development of the disease. From examination of literature, which is given at considerable length, he is not convinced that any cases of tabes are on record which can be attributed to trauma alone—injury may certainly hasten the development of the disease and the progress of a case. Collins’ statistics show that antisiphilitic treatment has no effect in preventing or delaying the development of tabes—indeed, these cases in which the treatment was carried out most fully developed tabes rather earlier than those in which the treatment had been desultory and incomplete. No new light is thrown on the pathogenesis of tabes—we do not yet know how syphilis causes tabes (*vid. infra*).

In the second paper, after giving notes of cases to illustrate the different clinical types of tabes, Collins discusses the *symptomatology*. The *initial* symptom, noted in 126 cases, was as follows:—Pain, 51 cases (*i.e.* 40 per cent.)—shooting pain in leg, 31 cases; difficulty of micturition or incontinence, 7 cases; loss of sexual power, 4; giving way of legs, 6; weariness of legs, 6; ataxia, 6; vertigo, 3; numbness of arms, 2; gastric crises, 6; rectal crises, 1; pruritus, 1; ulcer of foot, 2; ptosis, 5; diplopia, 7; weakness of knees, 6; numbness of feet, 5; numbness of leg, 5; partial paralysis of leg, 1; nocturnal emissions, 1; psychroesthesia of knee, 1. With reference to the comparatively small proportion of his cases in which pain was the initial symptom, Collins thinks that enquiry will often show the existence of earlier symptoms in cases where pain is stated to be the first symptom.

Analysis of his series of 140 cases gives the following results as regards the occurrence of the chief symptoms:—

*Pain* in 126 cases (90 per cent.), predominating in the legs in 84 cases, and present in the legs in 121 cases; *paræsthesia* in 103

(73 per cent.), affecting mainly the legs and trunk, in some cases very annoying in the domain of the fifth cranial nerve; *girdle sensation* in about a third of the cases; *disturbance of tactile sensibility* in 94 cases (67 per cent.), nearly as frequent on the legs as on the trunk over the fourth to the sixth dorsal segments—*anæsthesia* of the legs was present in 58 per cent. of the cases in which the sensory condition was specially noted; lessened keenness of tactile sensibility has little or no relationship to the duration of the disease, but *intensity* of disturbance has; *analgesia* in 82 cases (58 per cent.), relatively oftener than anæsthesia in the arms, most common in the domains of the ulnar (34 cases) and peroneal (43 cases) nerves; a zone of *analgesia over upper part of chest* in 53 cases (38 per cent.); *Biernacki's sign* in 18 of 42 cases (43 per cent.); *increased sensibility to cold* in 20 of 54 cases (37 per cent.); *impairment of deep sensibility* in 63 per cent. of 73 cases, mainly of legs; *hypotonia* in 41 of 57 cases (72 per cent.); *visceral crises* in 20 cases (14 per cent.), in 12 affecting the stomach, in 4 the rectum, in 3 the larynx, in 1 the intestines—in 6 cases, gastric crises were initial symptoms; *ataxia of gait* in 102 cases (73 per cent.); *Romberg's sign* in 117 cases (83·5 per cent.); *ataxia of upper limbs* in 48 cases (34 per cent.), much more frequent than statistics usually show; *knee-jerks absent* in 118 cases (84·3 per cent.), sluggish on one or both sides in 14 other cases, exaggerated in 2 cases; *Achilles-jerk absent* in 107 of 121 cases (88 per cent.), sluggish or present only on one side in other 10 cases; *elbow-jerk absent* in 19 of 44 cases (43 per cent.), exaggerated in 2; *plantar reflex* normal or lively in 59 of 81 cases—in no instance was the Babinski phenomenon present; *abdominal reflex* normal or exaggerated in 46 of 63 cases; *inequality of pupils* in 32 cases (23 per cent.), irregularity in 8 cases; *myosis* in 63 of 94 cases; *Argyll-Robertson pupil* in 107 cases (77 per cent.), no reaction of pupils in accommodation in 9 cases; *diplopia* in 32 cases (22 per cent.); *external ocular palsy* in 14 cases (10 per cent.), or, including cases with some degree of ptosis, in 21 cases (15 per cent.), much less frequent than text-books usually state; *nystagmus-like* movements in forced positions of the eyes in 21 cases (15 per cent.); *optic atrophy* in 19 cases (14 per cent.), in 6 leading to total blindness, in 4 the initial sign—in no case did the development of optic atrophy seem to retard or affect in any way the course of other symptoms, *e.g.* ataxia, pains, etc.; *bladder symptoms* in 78 cases (55 per cent.)—among the initial symptoms in from 10 to 20 per cent.; *impotency*, complete or partial, in 68 cases (50 per cent.); *arthropathy* in 7 cases (5 per cent.); *trophic lesions of soft parts* in 12 cases (8·5 per cent.); *muscular atrophy* in 18 cases—in 15 it appeared late, affecting more often the lower extremities and having the appearance of inactivity atrophy with no reaction of degeneration, while in 3 it appeared early, affected



the upper extremities and was accompanied by fibrillary contractions and reactions of degeneration, which pointed to involvement of the primary motor neuron; *insomnia* was noted in 21 cases; *vertigo* in 8; *disturbance of hearing* in 9; *glycosuria* in 3; *albuminuria* in 7; marked *loss of weight* in 37. In the writer's experience, *valvular disease of the heart* is not so common as Gowers and others maintain. In only five cases was disease of the aortic valves found, in two aortic aneurism, in 2 mitral regurgitation. In the majority of cases the *pulse-rate* was increased, but in no case were the clinical phenomena of Graves' disease found.

In the third paper Collins deals with the *morbid anatomy* and *pathology* of tabes, basing his remarks as regards the morbid anatomy upon a study of three cords. The lesions found in the cord, the posterior roots, the spinal ganglia, the nerve trunks and peripheral nerves, the cranial nerves and nuclei are described and figured. With regard to Clarke's column, he states that "it shows alterations in nearly every case of tabes. The cells themselves are not disordered, neither are their axones, but fibres that come from the posterior roots to ramify around the cells of Clarke's columns are seen . . . to have the typical appearance of degeneration."

As regards cases of tabes with muscular atrophy, "in the vast majority of such cases the cells of the anterior horns have been found intact. It is not improbable that a neuritis of motor nerves is responsible."

The changes described in the ganglia of the posterior roots are inconstant and irregular and, as a rule, trifling compared with the alterations in the posterior roots: "it is more than likely" that they are secondary either to disease of the pia or to alteration of the posterior roots in their intra-medullary and supra-ganglionic course.

The changes found in the peripheral nerves are similar in nature to those of the posterior roots, but "the process is in no way to be compared either in constancy or intensity with that of the posterior roots."

Collins reviews and criticises in some detail the various theories as to the *pathogenesis* of tabes: (1) The vascular theory. (2) The theory that the primary lesion is a degeneration of the posterior columns. Attention is directed specially to the recent view of Marie and Guillain that the initial lesion of tabes is a syphilitic affection of the posterior lymphatic system of the cord. (3) The theory that the primary and essential lesion of tabes is a degeneration of the posterior roots either primary or secondary to disease of the cells of the ganglia on the posterior roots or to compression of the posterior roots by a posterior meningitis (Obersteiner, Redlich)—also the view of Nageotte that the primary lesion is a syphilitic meningitis leading to a characteristic transverse neuritis

of the roots at the point of their emergence from the subarachnoid space. (4) The theory that the primary lesion is of the cells of the ganglia on the posterior roots, either organic (Stroebe, Oppenheim, etc.) or dynamic (Babinski). (5) The neuritic theory (Leyden, Goldscheider). (6) The theory of Thomas and Hauser that the essential lesion is a neuritis or rather a dystrophy which involves the entire peripheral sensory neuron, the lesion being predominantly, however, of the central prolongation of the cell.

The general conclusion arrived at by Collins is that "the evidence at hand tends to show that the essential lesion of tabes is an elective progressive degeneration, segmentary in type (in contradistinction to Wallerian), of the posterior columns of the spinal cord. The distribution of the degeneration therein corresponds with the intraspinal distribution of the posterior root-fibres," but it does not follow that the original lesion is in the posterior roots and that the changes in the posterior columns are secondary. While not admitting that any theory so far formulated adequately explains the pathogenesis of tabes, he thinks that that suggested by Marie and Guillain "would seem to be more in accord with what we know of the ætiology of tabes than any of the others. Adopting their theory, it is probable that the lesions of tabes are the results of the activity of a poison generated by syphilis acting primarily upon the intramedullary distribution of the posterior roots. The changes in other parts of the nervous system may be incidental to the existence of this poison within the system or they may be secondary to the disorder of nutrition superinduced in the entire peripheral sensory neuron by disease of such an important portion of it as that which is affected in tabes."

ASHLEY W. MACKINTOSH.

**ACUTE PRIMARY SPINAL OSTEOMYELITIS.** F. WEBER, *Deutsche (315) med. Wchnschr.*, May 7th, 1903.

WEBER records a case of acute osteomyelitis of a lumbar vertebra terminating in complete recovery. The patient was a lad of 15 years of age, of good previous health and with a good family history. His illness began three days before his admission to hospital with a rigor and pain in the back. On admission he showed the symptoms of a severe infection and complained of great pain in the back and abdomen. There was œdema over the lumbar spines and the spine in this region was exquisitely tender on palpation. The patient grew rapidly worse, and swelling extended from the 10th dorsal spine above, to the sacrum below. Fluctuation appeared opposite the 2nd and 3rd lumbar spines. The pain radiated along the nerves of the lumbar plexus, but there was no sign of involvement of the cord with the exception of some difficulty in micturition.

Operation disclosed a large abscess cavity among the muscles of the back, originating from the 2nd lumbar vertebra. The spine, the arch, and a transverse process, were found bare and infiltrated with pus. They were removed and a large quantity of pus escaped from the vertebral canal. The dura was intact. The result of the operation was good, but the patient still complained of pain in the right leg, and three weeks later another abscess appeared in the gluteal region which had evidently burrowed along the front of the spine and come through the great sacro-sciatic foramen. Two months after the first operation the patient had recovered completely. Cultures from both abscesses showed a pure culture of staphylococcus aureus. Weber believes that his is the only recorded case where the patient recovered after the development of a secondary abscess travelling along the front of the spine.

J. W. STRUTHERS.

**A CASE OF PROGRESSIVELY DEVELOPING HEMIPLEGIA  
(316) LATER BECOMING TRIPLEGIA, RESULTING FROM  
PRIMARY DEGENERATION OF THE PYRAMIDAL  
TRACTS.** CHARLES K. MILLS and WILLIAM G. SPILLER,  
*Journ. of Nerv. and Ment. Dis.*, July 1903, p. 385.

In this paper the authors give clinical and pathological details of a case of progressively developing hemiplegia later becoming triplegia, due to a primary degeneration of the pyramidal tracts. The patient, a man aged 54, was admitted to hospital in 1897, and was under observation till 1903, when he died. His family history was unimportant, he denied venereal disease, and had suffered from rheumatism six years before admission. He was a heavy drinker. His case was one of progressively developing right hemiplegia, in which the lower extremity was earlier and more markedly affected than the upper, and in which after several years the left lower extremity became paralysed, but to a lesser extent. The deep reflexes were all exaggerated and Babinski's response was present. The pupils reacted normally and there were no sensory symptoms. The patient suffered at times from severe intestinal and cardio-vascular symptoms, and eventually died from weakness brought on by these troubles.

The necropsy revealed serious pathological changes in the vessels and many of the viscera, chronic pleurisy tuberculosis with cavity formation, endocarditis and contracted kidney, with dilation of the aorta. The microscopical examination of the nervous system showed intense and long-standing degeneration of the right crossed and the left direct pyramidal tracts, the degeneration extending into the pons, but not into the left cerebral peduncle, also recent

degeneration of the left crossed and the right direct pyramidal tracts traced by the Marchi method into the lower part of the right internal capsule. No lesions, degenerative or focal, were found elsewhere in the brain or spinal cord. The case therefore was regarded as one of primary degeneration of the motor tracts, much greater and of older standing in the right crossed and left direct pyramidal tracts. The authors mention five similar cases which have been recorded, and also refer to ten cases of bilateral primary sclerosis of the pyramidal tracts which are vouched for as being indisputable evidence of such a condition.

T. GRAINGER STEWART.

**POST-HEMIPLEGIC HEMIATAXIA AND SUBCORTICAL CO-ORDINATION.** ED. CLAPARÈDE, *Rev. Neurol.*, July 15, 1903, p. 661.

CLAPARÈDE records a case of a woman of sixty-five suddenly affected with a slight left hemiplegia, in which the most marked symptom was the loss, not of motor power, but of the co-ordination of movements of the left arm. A careful examination showed that sensibility to and power of localisation of tactile and painful stimuli, the notion of position and the sensation of passive movement, were as good on the left as on the right side. The stereognostic sense was affected in only the very slightest degree, such as might be fairly ascribed to the ataxia of the fingers.

The author had in his thesis (*Du sens musculaire, à propos de quelques cas d'hémiataxie post-hémiplégique*, Geneve, 1897), arrived at a conclusion with regard to the sensory and co-ordinating paths similar to that formulated by Dejerine and Egger in the *Revue Neurologique* of April of this year, viz., that the sensory path before reaching the cortex of the cerebrum divides into two portions. One of these passes from the thalamus to the cortex, and subserves conscious sensibility. The other enters into relation with the neurones of the thalamus and corpus striatum, and is employed for automatic and reflex co-ordination, and is the only one put into action during our habitual movements. If the former of these two paths is alone affected (Dejerine and Egger), then there will be serious disturbance of superficial and deep sensibility without disturbance of co-ordination. If, on the other hand, the latter or optostriate path is involved, there will result inco-ordination without sensory disturbance.

To the question why the conscious sensibility, when it exists, does not re-establish co-ordination, Claparède gives the following answer:—"Co-ordination consists in putting simultaneously into action a large number of muscles, synergist and antagonist, the action of which is associated by experience, but not consciously.

The muscles acting are unknown, and the various combinations of their activity have not been registered in our conscious memory. When we wish to carry out a known movement, we represent to ourselves the object to be attained, the distance to be covered, and the movement is accomplished of itself, automatically, without our knowing in what order the elementary movements succeed each other. It is thus easy to understand why conscious sensibility may remain unaffected even with a well-marked ataxia.

ALEXANDER BRUCE.

**LARVAL EPILEPSY.** W. TSCHISCH, *Journ. of Ment. Pathol.*, Vol. iv. (318) Nos. 1, 2, and 3, 1903, p. 34.

THE author described the case of a man who during the night murdered four of his family, of which there were seven. All slept in the same room. Having committed the crime, the criminal was seen by a survivor to go out, urinate and vomit, and then to return to sleep. He was suspected of being insane, but during observation extending over some months no certain proof of insanity could be obtained, as he was a clever simulator. Prof. Tschisch then saw him, and discerned a peculiar metallic shine in his eyes, which he says is pathognomonic of essential epilepsy. This *blunt metallic shine* has been studied by him, and although difficult to describe, is easily recognised by personal observation. It does not exist in Jacksonian epilepsy, nor in subjects with cerebral tumours and fits. It is also absent in idiocy or imbecility with convulsions. Lustrous eyes are present in hysterical and maniacal conditions, but do not show the metallic gloss. This condition, the author maintains, is due to an intoxication, probably the same as that which causes the fit, and in support of his opinion he mentions the fact that he has observed that it is absent after a fit. Young subjects show it best, and old epileptics not at all. He notes the medico-legal value of this sign.

C. H. G. GOSTWYCK.

**CONTRIBUTIONS TO THE STUDY OF INFANTILE NEURASTHENIA.** (319) THENIA. CAPPELLETTI, *Rif. Med.*, No. 17, 1903.

It has been believed until recently that infantile neurasthenia rarely, if ever, occurred.

The author briefly refers to some cases already recorded, and then proceeds to describe two cases which he had observed himself.

1. A girl aged 11. Mother insane, uncle committed suicide.

Patient was not born at term, and was reared with the greatest difficulty, but after 7 health improved. Mentally fairly active, and of average intelligence.

Later began to be capricious, taciturn, dissatisfied and emotional. Frightened by attempted suicide of her mother a year and a half ago. A year ago began to complain of headaches and of vertigo. Says she often had noises in her ears; vision obscured; pain in abdomen and back; no palpitation.

She became very irritable, threw objects to the ground if opposed; applied herself less to school; preferred to stay at home. Said she did not feel capable of attending to studies. Lacked will to apply herself. Sometimes dull, sometimes no wish to go out or to play. Fear of doing wrong. Well developed; slight asymmetry of face. Nothing found by physical examination.

*Psychic Functions.*—Attention limited; would not come to decision; expression of indolence and doubt; very emotional and capricious; preoccupied with small things.

2. Boy of 12. Marked neuropathic family history; scrofulous diathesis; weak; small amount of energy. Psychically he is precocious, and still a boy is already a man, and that anergic; stays indoors; capricious, indolent, emotional.

Headaches; slight squint; teeth badly implanted and carious; ears large and projecting; vision weak on right side; sense of smell most acute, showing irritative condition of Schneiderian membrane of epileptic nature.

Intelligent; attention difficult; air of weariness and doubt; seems unable to come to a decision; emotional and somewhat irritable.

Neurasthenia is somewhat veiled in young children by the usual exuberance of that period of life.

The author distinguishes the following psychic facts as aids to the diagnosis of neurasthenia: (1) Defective attention; (2) taciturn disposition; (3) impaired memory; (4) diminution of will. And the following physical facts: (1) Pain in head; (2) vertigo; (3) insomnia; (4) myasthenic symptoms; (5) gastro-intestinal disturbances; (6) genital excitability (masturbation); (7) irritable heart; (8) vasomotor and secretory disturbances; (9) degenerative stigmata.

Many of these psychic and physical symptoms are seen in other conditions, but a careful examination should make the diagnosis fairly safe.

Underlying many of these symptoms and modifying them, is what the author calls the "state of doubt." This is always present, and often remains after the other symptoms have disappeared.

As to the character of this doubt the author suggests that



every deliberation of the normal man is the result of a ready selection between two antagonistic thoughts, and the short fight between the opposed thoughts constitutes in germ the state of doubt. This fight of ideas is therefore a normal process, but when prolonged, as it is in the neurasthenic, it leads to an alienation of the whole psychic life. This condition is met with in varying degrees, and often shows itself in the most trivial details—looking to see if a door is shut; reading and re-reading letters. At other times it causes a feeling of self-distrust, pessimism, diffidence, and spoils the natural happiness of life; causes the eternal preoccupation of the neurasthenic—mental stigma of degeneracy.

Hence their inability to complete anything taken in hand, to progress at school, and their desire to avoid other people.

Important to regulate the social and moral life of the patient.

R. G. Rows.

**SYMPTOMS OF MENINGITIS WITH PAROXYSMAL CHILDISH-  
(320) NESS IN AN HYSTERICAL PERSON.** DUPRÉ et CAMUS,  
*Rev. neurol.*, July 15, 1903, p. 657.

A ROBUST girl of 18 with a bad tubercular family history was the subject of this publication.

She was in a prostrate condition when she first came under observation with headache, vomiting and abdominal pain. The temperature rose, the headache became more severe and she developed convergent strabismus with myosis, slight unilateral facial palsy, positive Babinski's sign on one side and carinated abdomen. There was no lymphocytosis in the cerebrospinal fluid obtained by lumbar puncture. In a few days most of these symptoms became less marked, but hemianæsthesia and contraction of one visual field was then observed.

As the somatic symptoms disappeared she was confused, and her mental state was remarkably that of a child in both language, actions and sentiments. Her respiration was panting and irregular and her speech faltering. Babinski's sign was not obtained after the first few days, and her mental state was again normal in about a fortnight. She seemed annoyed and ashamed when shown evidence of her earlier childish conduct.

Four months later she again came under observation with more or less the same symptoms, but from these she again rapidly recovered. This second attack was attributed to the death of a sister from tubercular meningitis.

The hysterical nature of the meningeal symptoms is argued from the complete recoveries and the negative find in the cerebrospinal fluid. The fever was also regarded as hysterical. The

presence of Babinski's sign was attributed to a temporary affection of the pyramidal tract at its origin in the psychomotor area—*encephalopathie corticale*. From the recurrence of cerebral symptoms on the death of a sister from similar symptoms of organic origin, it is assumed that there was in the family an elective susceptibility of the meninges, a view hard to support by the mere coincidence of hysterical troubles with meningeal symptoms in the one sister and of tubercular infection in the other.

The interesting regression of the psychical state to an earlier stage has already been studied by one of the authors in other cases.

GORDON HOLMES.

**THE DIAGNOSTIC VALUE OF IRREGULARITY OF THE  
(321) CONTOUR OF THE PUPIL IN ORGANIC NERVOUS DISEASE.** J. PILTZ, *Neurolog. Centralbl.*, July 16 and Aug. 1, 1903.

DR PILTZ in this interesting paper gives first a historical outline of observations on this point, ranging from Baillarger, who noted the frequency of irregularity in the shape of the pupil in general paralysis (1851), to the observations of Joffroy and Schrameck (1902). The latter are of opinion that: (1) every change in the form of the pupil, every irregularity in the pupil (congenital anomalies of the iris and synechiæ being excluded), indicates one of three conditions, viz., general paralysis, tabes dorsalis, or central syphilis; and (2) that the diagnostic worth of this sign is not less than the Argyll-Robertson phenomenon, of which it is indeed the forerunner. As the result of his investigations Dr Piltz is in general accord with these views, and states as his experience that the sign is of very frequent appearance in general paralysis, that it has sometimes preceded any alteration whatever in light reaction, and that exceptionally in incipient general paralysis it has been the sole somatic symptom present.

For convenience sake Dr Piltz divides the changes into fluctuating and constant or permanent irregularities. In the first category there may be a transitory paresis of part of the iris, or an alteration in the site of the entire pupil. Such fluctuating changes may be observed in general paralysis, but the author has also noted the sign in katatonia (*dementia præcox*). In cases coming under the second category, the pupil may be angular, elliptical, pear-shaped, or combinations of these.

As regards the frequency of occurrence, Dr Piltz has found one or other of these changes present in 17 out of 38 general paralytics, and 18 out of 62 tabetics. Dr Piltz has been for some time of the opinion that these anomalies are due to defects in iridic innervation,



and, to throw light on the mechanism of these changes, has carried out a series of experiments on rabbits, cats and dogs, consisting in stimulation by the induced current of the ciliary ganglion and long and short ciliary nerves, after laying bare the orbit. He has by this means been able to produce changes corresponding exactly to the deformation of the pupil occurring in man, even to changes in position of the whole pupil. The operations and results are carefully described in the paper, and the conclusions are contained in the following condensed summary:—

1. The following pathological changes may be observed:—
  - (a) Temporary or fluctuating alterations of separate parts of the iris.
  - (b) Change of site of entire pupil.
  - (c) Constant irregularity.
2. All of these constitute frequent features of general paralysis, tabes dorsalis and cerebral syphilis.
3. Sometimes they are met in other organic nervous diseases, but only exceptionally (about 1 per cent.) in the sound.
4. Temporary changes occasionally appear in katatonia.
5. Irregularity of pupil contour sometimes precede the Argyll-Robertson pupil and have decided diagnostic value.
6. From experimental and clinical evidence it is probable that these changes are the expression of deficient innervation resulting from pathological changes in the particular fibres of the long and short ciliary nerves whose nuclei are affected.
7. Irregular motility of any separate part of the iris depends on a paresis (*sic*) of the corresponding fibres of the ciliary nerves.
8. Alteration in the site of the whole pupil is the result of a combination of such defects occurring in different fibres of both long and short ciliary nerves.
9. Constant irregularity is the result of a complete palsy of particular sections of the iris due in all probability to atrophy of the corresponding nerve cell.

R. CUNYNGHAM BROWN.

**THE REACTIONS OF THE PUPIL.** L. BACH and H. MEYER, *Von* (322) *Graefe's Arch. f. Ophthalm.*, lv. 3 and lvi. 2.

BACH, as is well known, is an advocate of the situation of the centre for the light-reaction of the pupil in the extreme upper portion of the cord. Some of his earlier experiments were open to certain objections, particularly that the resulting pain might have vitiated the conclusions as to the existence or non-existence

of a reaction, so they have been repeated under an improved method of narcosis. Without giving an extended account of these, it will be desirable to mention some of the conclusions.

1. Complete and even repeated section of the cervical cord several millimetres downwards from the fourth ventricle causes in the cat no change whatever in the pupillary reactions. At the moment there is dilatation, but this passes off in a few seconds, leaving the reaction as before.

2. Bilateral section of the medulla at a certain level just at the lower end of the fourth ventricle has as a result complete loss of the light reaction on both sides.

3. Section of the *right half* of the cord in a similar situation produces immobility of the *left* pupil. (Bach expresses considerable astonishment that the loss was not direct rather than crossed.)

4. If the medulla be freely laid bare, this is often sufficient of itself to cause loss of all reactions; and this is even more likely to be the case if strong stimuli have been applied as well. In fact, myosis and inequality of the pupils may be observed—a condition suggestive of tabes. This immobility of pupil, however, may be observed to give place to an active reaction if a section is now made through the medulla at or above the middle point of the fourth ventricle.

5. *Unilateral* section of the medulla at or above the middle point of the fourth ventricle causes the pupil reaction which had been sluggish or lost once again to become active on *both* sides.

According to their researches, there is an area of small dimensions close to the middle line and to the respiration centre at the spinal end of the fourth ventricle, which is of maximum importance for the light reaction of the pupil; it might be called the inhibition centre for the light reflex. The light reflex centre lies above (cerebral-wards from) this centre, not in fact below the corpora quadrigemina.

Further experiments seem to the two authors to show that in the medulla lies the (chief or only) centre for pupil-dilatation, and also a highly important centre for the inhibition of that centre. In addition to that in the medulla it seems highly probable that in other parts of the cerebro-spinal system several other centres exist for the dilatation of the pupil.

W. G. SYM.

**TWO CASES OF HEMIORANIOMA—HEMIHYPERTROPHY OF  
(323) THE SKULL.** BRISSAUD and LEREBoullet, *Rev. Neurolog.*,  
June 15, 1903.

THE authors publish two cases of what appear to be endotheliomata of the dura mater, which secondarily involved the skull and

so caused new protuberances, which in each case were limited to the one side. In the one case, a man now over 20 years, the prominence was in the parieto-frontal region, and had been growing slowly since infancy. Recently he had several generalised convulsions, with loss of consciousness, but no history of headache or vomiting, and no other objective physical signs.

The second patient in whom the cranial asymmetry was also visible since infancy was at the time of observation 31 years of age, and for two years had symptoms of intracranial tumour, headache, vomiting, consecutive optic atrophy with complete blindness, exophthalmos, and lapses of memory. There were no paralytic or sensory symptoms. The tumour was hard and limited to one side of the skull and forehead, and a small mass lay in the palatine arch. Death resulted from cerebral symptoms.

At the autopsy both dura and bone were found very thickened and adherent to one another, and from the inner surface of the dura several masses of tumour protruded into and compressed the brain. Even the latter were partially calcified. The microscopical diagnosis was angiolithic sarcoma.

GORDON HOLMES.

**ASYMMETRY IN AN INFANT: OR, CONGENITAL HEMI-  
(324) HYPERTROPHY. A. HYMANSON, *Arch. of Ped.*, June 1903,  
p. 428.**

A GOOD and very complete instance of this rare malformation is described by Hymanson. The patient was a male child who was under observation from the sixth to the eighteenth month, during which period the hypertrophied side grew rapidly, the normal side somewhat slowly. As is usual, the right side was the seat of the overgrowth, but, compared with most other cases, the hemihypertrophy was very widely diffused, involving head and face, thorax, abdomen, arm and leg. It is, I think, the most complete case recorded up to the present; in most of the so-called "total" hemihypertrophies it has not been possible to demonstrate asymmetry of the pelvis and abdomen. The heredity is important only in the absence of all history of deformity. The hypertrophy was noticed at the sixth week. Measurements of all parts of the right half of the body are greater than those of corresponding parts of the left side. Usually in such cases the more distal portions are most affected, but judging from the photographs this was not so in Hymanson's patient. The hair on the left (normal) side of the head was coarser and less curly than on the right, the eruption of teeth was not earlier on the larger side, the child was stronger in the left normal than on the right hypertrophied side, and there were no nævi or angiomata. In these four respects the case is an exception to the usual rule in this condition. The interesting

observation (hitherto, so far as I know, unrecorded) was made that in ordinary eclampsia the convulsions were most marked on the right side. The various theories of the pathology are briefly discussed, the view favoured being that of congenital lesion of the vasomotor centres with vascular stasis. We are, however, quite in the dark as to the cause of hemihypertrophy, and it is possible that its origin may not always be the same. Hymanson quotes England's recent case of facial hemihypertrophy, in which neurofibromata involved all the cranial nerves, as supporting the nervous origin of the condition. To show how great diversity of opinion exists, some of the theories which have been suggested may be mentioned: premature vice of the middle layer of the blastoderm; partial intrauterine strangulation of the affected member; inherent tendency of the tissues to appropriate excess of nutriment; lymphatic defect, analogous to elephantiasis; defect of the muscular coat of the arteries; nervous origin. Hymanson's case is of great interest; one would, however, have liked to have learned from skiagrams whether the overgrowth affected the bones, or was limited to the soft textures.

J. S. FOWLER.

### PSYCHIATRY.

**THE PATHOGENESIS OF THE SPECIFIC DELUSIONS OF (325) GENERAL PARALYSIS.** A Contribution to the Investigation of General Paralysis by Experimental Psychology. A. WIZEL, *Neurol. Centralbl.*, 16th July 1903, p. 668.

As its sub-title indicates, this paper gives the results of some experiments to test the basis of the delusions which are most commonly met with in general paralysis. The author believes these to be characterised by their extravagant exaggeration and uncontrolled hyperbolism. In the paranoiac, on the other hand, they are usually confined within the bounds of possibility. This distinction does not rest on the amount of intellectual impairment which is present, for in general paralytics their most expansive delusions are most marked in the earlier stages, when the intelligence is less affected than it is at a more advanced period in the disease. The experiments were designed to test, in general paralytics, the sense of time and of space, as it appeared that their delusions were largely based on these factors.

The methods employed in the investigation were very simple. A metronome was set to beat 200 times a minute, and kept going for various periods, which the person under investigation was asked to estimate. The times chosen and their sequence were  $\frac{1}{2}$ , 2, 1,  $\frac{1}{4}$ ,  $1\frac{1}{2}$ , and 3 minutes. In five healthy persons who were

tested in this way the average error was about 55 per cent., the estimate of time being, almost without exception, too long. In five slightly demented patients the average error was about  $2\frac{1}{2}$  times as great, and in the same direction. In one general paralytic the estimate was absurdly too great. Thus,  $1\frac{1}{2}$  minutes were thought to be 40 or 60. Similar results were obtained in the experiments on the sense of space, though the error was much larger.

The conclusion drawn from these facts is that the general paralytic has lost all mental representation of time and space. Consequently all ideas involving these representations are subject to no rational control, and are invariably estimated wrongly. The same is probably true with respect to all their other mental processes, and so their typical expansive delusions may be explained.

JAS. MIDDLEMASS.

### TREATMENT.

#### REMARKS ON THE OPERATIVE TREATMENT OF CHRONIC

(326) **facial palsy of peripheral origin.** CHARLES A. BALLANCE, H. A. BALLANCE and PURVES STEWART, *Brit. Med. Journ.*, May 2, 1903.

In this valuable article the authors' main contention is that, in future, facio-hypoglossal anastomosis should be adopted in place of facio-accessory union.

The first portion of the paper contains notes of six cases operated on by the latter method, and of one case where facio-hypoglossal anastomosis was the operation adopted. If, as they have previously demonstrated ("The Healing of Nerves," 1901), regeneration occurs in the distal segment of a divided nerve, no interval of time, according to the authors, ought to be too long for attempted reunion so long as any muscle fibres survive which can be innervated by the regenerated and reunited nerve. Thus, in one of their cases, paralysis had existed for almost three years, and they see no reason why this limit should not be greatly exceeded.

The cases suitable for operation are especially those of palsy due to traumatism or to involvement in the Fallopian aqueduct by a suppurative otitis media, in which the paralysis has existed for six months or more without any sign of improvement. The special reason why facio-hypoglossal anastomosis is to be preferred to facio-accessory anastomosis is, in the opinion of the authors, the greater prospect of obtaining movement of the facial muscles unassociated with movement of other muscles. So far as their experience goes, they have not observed in their facio-accessory cases any independent movement of the face unassociated with

movements of the trapezius and sterno-mastoid. This they consider a very important drawback to the operation.

In this connection, however, it is only right to mention that Kennedy (*Brit. Med. Journ.*, May 16, 1903) draws attention to the fact, that although on sudden elevation of the shoulder a marked spasm of the face occurred in his case (referred to by the authors), voluntary movements of the face were apparently perfectly dissociated. He is further inclined to think that the possible limit of recovery has not yet been reached in the authors' cases.

Sufficient time had not elapsed in the authors' case of facio-hypoglossal anastomosis to allow them to judge whether dissociation was greater after that operation. On theoretical grounds, however, they are strongly of opinion that it should be. Dissociation of movement being a matter of education of the cortex, it seemed likely that if some healthy nerve were selected for anastomosis with the facial, whose cortical centre was nearer the face centre, the prospects of dissociated facial movement would be much greater. In the case of the shoulder, the centre is at some distance, namely at the junction of the upper and middle thirds of the ascending frontal gyrus. In the case of the tongue, that centre and the face centre overlap one another. Further, the movements of the tongue were closely associated with those of the lips, and it was more than probable that the facial nerve derived its lip fibres from the hypoglossal nucleus.

In a letter to one of the authors, quoted in the paper, Professor E. A. Schäfer made the suggestion that the glosso-pharyngeal was also a suitable nerve for anastomosis with the divided facial, on the ground that its motor nucleus is much nearer to, and is apparently serially homologous with, the facial nucleus. The authors, however, think that the small size and relative inaccessibility of the glosso-pharyngeal would make the operation one of extreme difficulty.

As regards the actual method of union in cases of nerve anastomosis, the end-to-side operation is preferred. A longitudinal incision is made in the sheath of the spinal accessory (or the hypoglossal) exposing the nerve fibres, a short incision being also made in sheath of the distal segment of the divided facial. The end of the facial is then fixed to the wound in the spinal accessory (or hypoglossal) by fine silk, the adjacent edges of the *sheaths* only of the two nerves being taken up in the stitches. An invariable immediate result of the facio-accessory operation is a temporary palsy of the sterno-mastoid and upper part of the trapezius. This clears up completely in all cases. In the case of facio-hypoglossal anastomosis, examination about three weeks after the operation revealed that considerable atrophy of the tongue on the affected side (the right) had occurred, and that the

tongue was protruded to that side. The facial muscles were, as usual, being treated by galvanism, but no improvement had, of course, as yet been noticed in them. The prognosis after operation is less favourable after suppurative conditions than after traumatism.

A. A. SCOT SKIRVING.

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## Reviews

**LA MENTE DI EMILIO ZOLA.** L. BIANCHI, *Ann. di Neurolog.*, Anno. xx., 1902, fasc. vi.

THIS paper, read at a meeting of the Neapolitan Democratic League, held in commemoration of Emile Zola, is in reality a psychological study of the mind of the great novelist. What, asks the professor, is the reason of the great disparity in the judgments expressed upon Zola and his works? The explanation is that the writer and his works may be viewed from very different standpoints: it may be that of the artist, the psychologist, the sociologist, the biologist or the politician. When a man reveals his thoughts in works that set in vibration the minds of men everywhere, that arouse the deepest sentiments of the individual and the community, that go beyond the customary circle of the human mind; when these works touch upon politics and religion, the two great factories of the human passions, and when new dominions of science are laid open to the popular intellect, and new springs of life pour out new aspirations, one can understand the upheaval of the collective consciousness, from which some old support has fallen. Zola's work must not be judged only in its particulars, its literary form, its style, its episodes, the scenes portrayed, the colouring, the harmony of the phrase, or the individual parts of the great structure conceived by his mind; it must be judged especially in its entirety, its plan, its architecture, its development and its finality. Zola portrays life such as it really is, and he does so by the methods of modern biology. He has made a minute dissection of the social organism and has investigated its most hidden components; he has made very delicate microscopic sections, has coloured them with his art and has projected new and stupefying images upon the popular consciousness. He does not write to give delight, because when he comes upon the disagreeable and repugnant he takes it without scruple, throws it into his intellectual crucible and clothes it in art. An unprejudiced and sincere realist, he hides nothing of life, what it is or what is in it. He was, perhaps without wish-

ing it, a pathologist. In "L'Assomoir," "Terre," "Bête Humaine," "Guerre," "Germinal," in all the collection of the Rougon-Macquart series, there are sublime pages full of life surprised in its efflorescence, its triumphs, and in all the grades of its decadence and perversions.

Professor Bianchi suggests that possibly the strong inclination exhibited by Zola in his youth for the study of the natural sciences, or that heredity through his father who, as an engineer, must have studied natural science as well as mathematics, explains the biological basis of the romances. Zola does not confine his mind to historical and religious episodes, to the sensations, emotions, character, tendencies, costumes of a past epoch, or the aspirations of the individual, family, nation or race, building from them a fine literary edifice, richly furnished with pleasing phraseology, gorgeous in its æsthetic harmony, containing a solitary passion that stirs our consciousness and is deposited in it. This is one form of realistic romance, but it is not Zola's. Zola is a fine, acute, sincere and happy observer of modern life in all its forms, all its attitudes, all its commotions, its aspirations, its joys, its griefs, passions, instincts and all its forces. It was his desire to hold an inquest on the social life of his time, and the results of his enquiries are found in the Rougon-Macquart series and in "Paris." In these works of art he portrays man entirely as he finds him, not in the fog in which the artist paints him. He strives to show how example, circumstances and heredity are the great factors of human conduct, producing joy or grief, wealth or poverty.

Professor Bianchi brings under review the various types of humanity described in the different romances. He remarks how no type of a modern well-bred woman is brought upon the scene—no familiar feminine incarnation of virtue, because such a type would have been wasted in the design of this work—yet who can deny the reality of the types that we do find? Maurice Barré attributes to the mind of the romancer the obscenity that fills his romances, while Bianchi holds that it was his ideal of regeneration of a people whom he saw on the downward path of degeneration and decadence. The types that Zola brings before us represent all the evolutive and degenerative gradations of man in the historical period of the nation, and serve as a condensed treatise of teratology and social pathology. The whole Rougon-Macquart series represents a genealogical tree commencing with Adelaide Fouque and ending with Charles Rougon, and in which we find also Gervase, Nana, Lantière, Coupeau and Doctor Pascal—all the forms of life, vices and paranoic talents, weaknesses, eccentricities and miseries engrafted on all the social gradations, making degenerative strides from the political representative to



the washerwoman and prostitute. Thus the artist symbolises the human degeneration that commences with a mental affection of the progenitor, and ends in the complete extinction of the stock. Herein lies the whole social pathology: The neuropathic heredity gives the degeneration, alcohol and the abuses of life aggravate it; the degeneration gives lassitude, lassitude gives sensuality and alcoholic inebriety, and the value of the life is lowered.

In "Lourdes" again, Zola's ideal of regeneration is more apparent. The marvellous pictures he draws of the various invalids and their diseases remind us of pages in the best medical works. Zola examines the mystic side of life and successfully reveals to the people the natural mechanism of the cures. He institutes in art, as histologists have done in science, a war against mysticism, which every day is retreating before the penetrating and discerning forces of the human understanding. With "Lourdes" he inaugurates a vast scheme of social therapeutics and prophylaxis with the object of correcting the evils that afflict France. The limitation of family appeals strongly to Zola, and in "Fecondite" he attacks this violation of the natural laws and the tendency to infanticide now prevalent in France.

In "Paris" he assumes rather the character of the teacher of ethics and social reformer. This tendency is accentuated in "Travail." He formulates clearly the conception of a religion of humanity, and in fulfilling this design he abandons the naturalistic rigidism of the Rougon-Macquart series and becomes an idealist. Bianchi explains how this new rôle is the result of a natural process of evolution in the mind of Zola. He discusses the various intrinsic qualities of Zola's intellect and the different influences in the environment that give to Zola's mind its characteristic orientation. He shows how, from the nature of things, it was unavoidable that Zola should embrace the naturalistic doctrine of morals in the sense of Lewes, Spencer and others, and should set about freeing the religious mind of the people from the old mass of prejudices that science had already victoriously torn from mysticism.

But in the process of mental evolution in Zola a just proportion has not been maintained. The biologist is diminished and imagination is uncurbed. It is impossible to effect in a few generations the transformation of types such as those of Mebotte and Nana into types like Luc and Jourdain; it is impossible to obtain an equal measure of stimuli operating on each individual, and hence an equality of sensibility for pleasure and pain, and a kindly quiescence of the instincts and desires of individuals and peoples. Education and legislation may strive after this new religion of justice and truth which every day makes itself felt more strongly among us, but nothing can force or violate the law

of evolution of the mind; nothing can ever assume higher importance than the *quantity* and *nature* of the individual force. The formation of a consciousness of universal love may require more time than the formation of the actual human body.

Zola, after dissecting modern life in every detail, generates discontent in his mind, and with this a desire for better. He is led away by unrealisable ideals. There is evolution in his thought, revolution in his nature. This is the outcome of an ideo-emotional exaggeration characteristic of the man and the race. The people of the south acquire a stronger excito-motor force with a tendency to immediate action. Those in the north move more slowly but they utilise scientific truths for their material well-being, and in the struggle for existence they are victorious. Without question, Zola's vision of a perfect society such as he describes, is an idealistic creation that surpasses the limits within which he had till then confined himself. But, asks Bianchi, do these and other faults diminish the great and resplendent figure of Zola? Where shall we place him in the scale of intellects? Some have styled him a degenerate, others exalt him to the rank of genius. He is not a degenerate. To give expression to what the senses and intellect receive from the environment, with the view of awakening the collective consciousness to the tendencies and character of the people, is not degeneration.

Do we shrink with repugnance from the abomination and corruption of the miner, the filth of the laundry, the feminine libertinism flavoured with alcohol and the immodesty of Teresa Raquin and Virginia, and yet forget the sensuality of southern Europe? Do the obscene expressions of Nana, Ragu, Coupeau displease us merely because they pass through the brain of Zola, —those same things that we hear from the mouths of low women on the streets? Zola appears excessively sensual because he unconsciously portrays the intonation of the surrounding life, incarnating in his work the fundamental principles to which it gives expression, viz., that life is the complex product of heredity and environment.

Nor are the nervous temperament and the highly-developed sense of smell signs of degeneration in Zola. Though nervous disturbances may give rise to degeneration in the offspring, near or distant, it is not legitimate to generalise the idea of degeneration. Again, if man, who is the synthesis of all animal nature, could add to the strength of all the other senses the acuteness of smell of the dog for all substances while the dog possesses it for certain substances only, he would be more perfect. Some men are especially visual, others auditory, others tacto-motor or olfactory. Zola was neurotic but not degenerate. Is he even a pessimist? Professor Bianchi answers No, and points for proof to

the reintegration of the moral consciousness and dignity seen in Elena and Doctor Deberle; to the love of Teresa Raquin; to the scientific light that is shed upon mysticism in "Lourdes"; to the titanic struggle of Luc for the realisation of his ideal of love and well-being among the labourers; to the conception of life that we learn from the lips of Doctor Jourdain. The pessimist, says Bianchi, is also a negativist, while the whole work of Zola is a solemn affirmation of life and science.

Is he a genius? Is Dante, is Michael Angelo, is Leonardo da Vinci, is Darwin, is Volta? Zola resembles none of those lights of humanity. But must the mind always reach the same height in order to assume the dignity of genius? Must genius always be accompanied by the degeneration that we call epilepsy, paranoia, or hysteria? What is to be the standard of comparison? How can we compare Wagner to Stephenson, Shakespeare to Volta, Cromwell to Christopher Columbus, Darwin to Manzoni?

There are sensory geniuses, intellectual geniuses, and mechanical geniuses; how can we put them together? Shall we call Wagner a genius because of his mental derangement and not Stephenson through whom the value of life has been enormously increased? Shall we seek to find degenerative notes in Shakespeare, a realist who sums up the human thought and sentiment, the past and the future, who lives to-day and will live; and shall we refuse to recognise genius in Volta because in his disputes with Galvani he succeeds in mastering one of the most marvellous forces of matter, through which the face of the world has been transformed in less than half a century? Shall we haply grant the title of genius to Cromwell who had the hallucination of a beautiful lady prophesying that he would be a great man in the State, and refuse it to Bismarck who, with an extraordinary power of calculation, and without hallucinations or neuroticism, played his own game in the East and in the West and raised Germany to an unforeseen height of influence in the world.

Professor Bianchi does not discover any notes of genius in the individual parts of the various figures of Zola. In science he is not a genius, for he exhibits no originality of research; nor in art, for there is no originality of method. His genius, according to Bianchi, is in the artistic conception of an immense design representing modern life and especially its pathology; in the marvelously complicated architecture of the whole romance of life and in the artistic conception of life tuned to the grief that the bent of the times caused him. In the manifold forms and directions of activity of the human understanding one man among many may rise to a height never reached by any other and indicate to humanity a new adaptation, and in this we perceive a kind of genius. Again, see Zola in the rôle of accuser, arrayed against the evils in the official-

dom of France. He finds that the same evils that have root in the people of "Assomoir," "Germinal," and "La Bête Humaine," extend, like branches of a great tree, into the army, the magistracy, and the government. It is neither friendship for Dreyfus nor personal interest that determines his action. Sacrificing everything he confronts the constituted bodies of the State in the cause of truth and justice, and with extraordinary courage leaps to a height never before attained by man under similar circumstances. That courage and power represented the synthesis of all the healthy part of France and of civilised humanity. Zola transformed it into individual courage and conquered in the name of a universal principle. Therein lies the apotheosis of his work, another aspect of genius, the genius of moral force, the beneficent genius that sums up the past and the future, making clear to humanity the path of civil progress, that of truth and of justice.

J. H. MACDONALD.

**TABES AND MENTAL DISEASE (TABES UND PSYCHOSE).** A Clinical Study by Dr R. CASSIRER. Berlin, S. Karger, 1903. Pp. 124.

THIS interesting monograph is devoted to an important subject which has recently received considerable attention at the hands of several well-known specialists. The mental phenomena found in cases of tabes are here very fully described and discussed. As was to be expected, a large amount of space is devoted to the investigation of the relation of tabes to general paralysis. The author states that it was Westphal who, in 1868, described the occurrence of spinal symptoms in some cases of general paralysis, a disease which had formerly been regarded as a purely cerebral affection, and who gave an account of the spinal changes which he found to accompany these symptoms. He came to the conclusion that the cerebral and spinal changes are not due to the propagation of the diseased process from the one to the other, but are to a certain degree independent. He also acknowledged the very close relations of general paralysis to true tabes. Cassirer enters more fully into this question and discusses how far the changes in the posterior columns in general paralysis are to be looked on as truly tabic, and what relations exist between true uncomplicated tabes and general paralysis with posterior column affection, as well as other forms of paralysis. This question is looked at from three points of view, the etiological, the pathological, and the clinical. As to the first, he acknowledges that syphilis is the preponderating *conditio sine qua non*. This, however, does not mean an identity of the two diseases. The influence of heredity and of stress is

considered at this point. As to the former, it is stated that opinion is coming round to regard it as of more importance than was formerly attributed to it, while the influence of the latter is undoubtedly potent in both cases. He concludes that we get little assistance in solving the problem from the etiological point of view. The same is true, he considers, with respect to pathological anatomy. In this he differs from several other authorities, though he takes refuge in saying that in neither disease has the true pathology been arrived at.

Coming to the clinical evidence, he inclines to the conclusion that this does not favour the view of their identity. He lays stress on the differences in the course of the two diseases. It seems, however, only reasonable to expect that such differences would be found. Most cord diseases are slow in progress, whereas the reverse is true of most of those affecting the cerebrum. In the latter, also, the influence on other bodily systems is very much more powerful, and there are exceptional instances in both cases. Tabes may run a very rapid course, and general paralysis may be very prolonged, nearly thirty years being recorded in one instance. Both, too, are subject to remissions, and even to apparent cure. Another point that should not be forgotten is embodied in the observation of Hughlings Jackson, that "half of the symptoms of nervous diseases are due to the unbalanced action of healthy structures."

Having then to his satisfaction disposed of the question of tabes and general paralysis, the author turns to the subject proper of his thesis: the mental symptoms observed in cases of tabes. He thinks these are for the most part fortuitous, as they are by no means numerous. Most tabetics suffer from their disease without any great affective disturbance. Some are happy and hopeful, more are slightly depressed, while some show neurasthenic symptoms. Some are unaffected mentally from beginning to end. Some, on the other hand, show signs of enfeeblement of mind. As the result of his own experience the author concludes that any form of mental disease may occur in a tabetic, though some are much rarer than others. He turns his attention first to paranoia, and relates several illustrative cases. Hallucinations and delusions are not uncommon. The latter, especially, are to be expected from the variety of sensory disturbance that may occur, these forming a ready basis for disordered mental action. Hysteria is also occasionally met with. The author notes that in his experience optic atrophy is more often associated with psychosis than other forms of tabes. Mania, Melancholia, *Folie circulaire*, and other distinctive forms of mental disease are not often met with.

In the section on diagnosis most attention is paid to its differentiation from general paralysis. This is acknowledged to be

often a matter of the greatest difficulty, and usually can only be settled after repeated and prolonged observation. The progress of the disease and the degree of mental impairment are looked upon as the best factors by which to arrive at a correct diagnosis. Alcoholic factors, though difficult to discriminate at first, are usually determinable after a time. Difficulty is also experienced when tabes is complicated with other syphilitic processes leading to mental symptoms. In such cases the exclusion of general paralysis may be almost impossible.

The book closes with a list of references bearing on his subject which is complete and most useful. There is, however, no index.

JAS. MIDDLEMAS.

**RESEARCHES UPON THE PAIN-SENSATION.** SYDNEY G. L. R. ALRUTZ. Upsala, 1901.

THIS is a treatise of some 130 pages, the author being a Licentiate in Philosophy at Upsala. It is a critical digest of work done upon the subject in question, together with an account of the author's individual work. Successive chapters deal with the following subjects:—Pressure-points, or *areae*; pain-points; the dual pain-perception (as described by Goldscheider); pain, the nerves of pain, and tones of sensation; perceptions of tickling and itching; the different qualities or characters of the pain-sensation, as induced by different modes of irritation, such as chemical (chloroform, ether, menthol) and thermal. The plan followed in each chapter is to give, in the first place, a historical survey of work done, upon which follows an account of the author's own researches; and, lastly, comes a discussion upon the subject-matter of the chapter. At the end of the pamphlet the bibliography of the subject is given: many of the authors mentioned are copiously referred to in the text.

The impression gained from a perusal of this pamphlet is that considerable diversity of opinion obtains upon the subject of sensory points and sensation. The author is able to throw light and to make a definite statement here and there, but upon the whole his researches do not appear to be conducive to greater certainty.

As regards the method employed for testing pain and pressure sensations, he employed Thunberg's glass-filaments instead of the hair used by v. Frey and others, and gives grounds for his preference. A series of glass-filaments of different degrees of "strength" was employed, tested according to a method which is described.

Whilst the scarcity of definite conclusions and the absence of dogmatic statement make it difficult to review a work of this sort, it should be said that the author shows himself to be very well



informed upon his topic, and to be possessed of critical and independent judgment. The pamphlet cannot fail to be instructive to those interested in the subject of sensation in its various forms.

E. GOODALL.

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# Review of Neurology and Psychiatry

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## Original Articles

### THE MORPHOLOGY OF THE HUMAN CEREBELLUM.

By G. ELLIOT SMITH, Cairo.

THE unsatisfactory nature of the commonly-adopted mode of subdividing the human cerebellum is so generally admitted that no apology is necessary for attempting to modify it so that it may have some definite morphological meaning.

The objects of these notes are to call attention to the chronological order in which the fissures make their appearance in the developmental history of the human cerebellum and to compare the resulting subdivision of the organ in Man with that of other mammals. In order to emphasise the homologies of the various regions I shall employ the nomenclature which I have recently adopted (1) in the case of the other Mammalia, in which it is impossible to apply the terms commonly employed in treatises on Human Anatomy.

In addition to the information which I have been able to acquire by personal observation, I shall make free use of the rich storehouse of facts graphically presented in the great monograph of Retzius (2) and of the data collected by Kuithan (3) and Stroud (4 and 5).

At an early stage in development two rudiments derived from the dorsal laminae (Flügelplatten of His) invade the roof of the fourth ventricle and unite in the mesial plane to form the primitive cerebellar bridge [Serres, 1815; His, 1892; Schaper (in Teleosts), 1894; Kuithan, 1895; and Stroud, 1895]. From the outset the lateral parts are much plumper than the mesial

region, and the statement that the so-called "vermis" is the first portion of the cerebellum to be developed is a pure fiction.

At the end of the second month, when the embryo is about 6 cm. long, we find that the extreme caudo-lateral angle of the cerebellum has been marked off as the flocculus by a floccular fissure, which cuts into the lateral margin and gradually extends in a mesial direction parallel to the caudal (ventral) margin of the cerebellar rudiment (compare Stroud, 4, plate v. figure 56). This rudimentary flocculus is directly continuous with the tuberculum acusticum, which lies immediately to its caudal side. At the same time or slightly later (*i.e.* at about the commencement of the third month) the nodulus becomes mapped out (figure 1) at the caudal (lower) part of the mesial region by the development of the post-nodular fissure (compare Retzius, Taf. iv. figures 2, 3 and 4).

It often happens that the floccular fissures (which are being prolonged mesially) fuse with the post-nodular fissure so as to cut off the whole caudal fringe of the organ, which is composed of the central azygos nodulus, the flocculi at the extreme lateral angles and two connecting bands, which may be called *alæ noduli* (compare Retzius, Taf. i. figure 34). None of my specimens enable me to fix, even approximately, the date of the first appearance of the parafloccular fissures; but, according to Stroud's illustrations, a slight furrow parallel to the floccular fissure is already present in the third month marking the limit of the paraflocculus (the insignificant flocculi secundarii of Henle, the flocculus accessorius of other writers). In man the paraflocculus is always insignificant, but in all other mammals, excepting only the Gorilla, the Chimpanzee and the Orang, it becomes a large and very prominent lobule; in the Sirenia the two paraflocculi may form more than one-third of the whole cerebellum.

Towards the end of the third month (in embryos about 10 cm. long) another furrow is revealed in a mesial sagittal section (figure 2). This is the *fissura prima* (*sulcus superior anterior*, *sulcus praeclivalis*, *sulcus primarius*, *sulcus furcalis*), which becomes in the adult the deepest furrow crossing the mesial plane. (For the earliest form of this fissure, see Retzius, Taf. iv. figure 3, and Kuithan, figure 20 (*s.p.*), p. 33.) This fissure rapidly extends laterally, and may be seen on the lateral



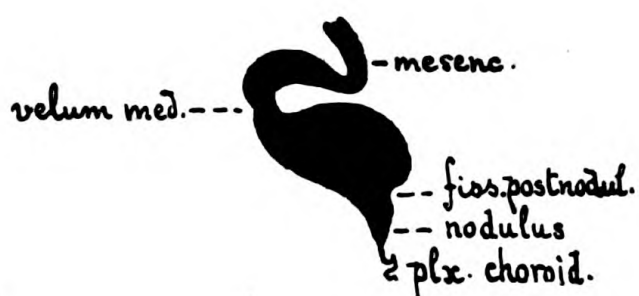


FIG. 1.



FIG. 2.



FIG. 3.



FIG. 4.

Figures 1-4. Diagrams representing mesial sagittal sections through the foetal human cerebellum in four different stages.

Figure 1. Embryo 9 cm. long×6.

Figure 2. Embryo 11 cm. long×6.

Figure 3. Embryo 12 cm. long×6.

Figure 4. Embryo 14 cm. long×6.

α—velum medullare anterior (superior).

β—lingula.

γ—pars praeculminata.

fiss. a—fissura intraculminatus.

aspect of the organ proceeding horizontally forward toward the middle peduncle (figure 5).

I have adopted this fissure (which is constant in all mammals) as an interlobar dividing line, calling the region

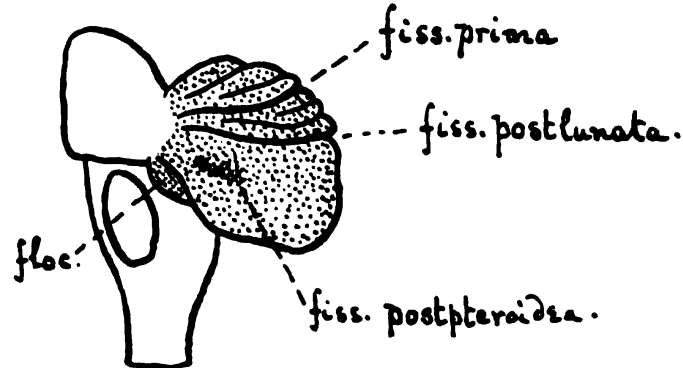


FIG. 5.

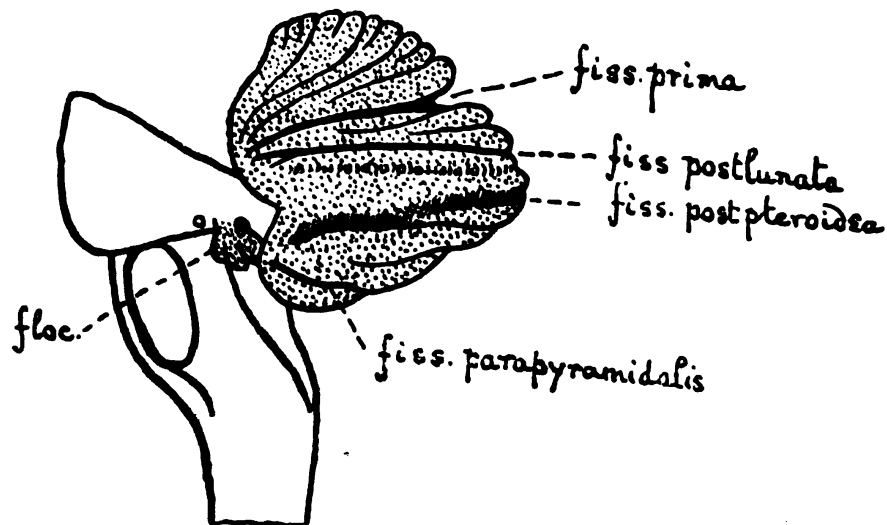


FIG. 6.

Figures 5 and 6. The left lateral aspect of two foetal human cerebella.

Figure 5. The cerebellum of a Fellah ♀ foetus at the beginning of the fifth month, 14 cm. long×2.

Figure 6. The cerebellum of a Soudanese ♂ foetus at the end of the fifth month, 26 cm. long×2.

above (cephalad of) it the lobus anticus, and the region immediately below it the lobus medius.

At the beginning of the fourth month (figure 3) two other furrows are usually revealed (in a mesial sagittal section) in the interval between the fissura prima and the fissura post-nodularis

(compare Retzius, Taf. iv. figures 6-9 ; Kuithan, figure 20 ; and Stroud, figure 64). That nearest to the nodulus is the fissura secunda, which indicates the posterior (dorsal) limit of the uvula and also of the lobus posticus (composed of the nodulus and uvula). The other fissure is the suprapyramidal, and the area included between it and the fissura secunda is the pyramid. In the smaller and more primitive Mammalia the fissura secunda develops much earlier than the fissura suprapyramidalis ; in fact, in several genera the latter is not present even in the adult. But in some of the larger mammals, including man, it often happens that the fissura suprapyramidalis is the more precocious and deeper of the two furrows (figure 4 ; also compare Kuithan's figure 20). This, however, is not invariably so ; for it sometimes happens that the phylogenetically older fissura secunda develops before the suprapyramidal fissure in the human brain. Great confusion has recently been introduced into the discussion of this subject by certain writers who imagine that homologous fissures develop in the same chronological sequence in various mammals. But just as in the cerebrum the sulci do not follow the same order of development in different mammals—for example, the recent sulcus centralis in man usually develops before the (phylogenetically) much older inferior frontal and intraparietal sulci so in the cerebellum fissures which are called into being by the precocious expansion of special areas in the larger mammals may make their appearance in development before some of the phylogenetically older fissures. A striking example of this will be seen (*vide infra*) in the case of the post-tonsillar fissure. The identity of the mesial parts of the cerebellum in various mammals may be readily recognised by studying the behaviour of their lateral connections, and we are thus able to check and correct the otherwise misleading indications derived from the mere study of the development of the pattern revealed in the mesial sagittal sections of series of embryos. The neglect of this obvious precaution on the part of certain recent writers has led to some very astounding conclusions, the patent absurdity of which does not seem to have presented itself to their authors.

Early in the fourth month the lobus anticus becomes subdivided by a fissure (figure 4) which pursues a course parallel to the fissura prima ; and in a short space of time this lobe becomes

split up into numerous lobules, while the other lobes of the organ are still smooth. In some mammals this precocious development of the anterior lobe is even more pronounced, for several of its subsidiary fissures may make their appearance even before the fissura secunda and the fissura suprapyramidalis are developed. The exact mode of subdivision of the anterior lobe in the human cerebellum is subject to a considerable amount of variation. In most cases it becomes split up into four lobules, which we might call (from before backwards) the first, second, third and fourth. The first lobule is the lingula, the second is the so-called "lobulus centralis" [the pars praeculminata of my earlier memoir (1)], and the third and fourth lobules together represent the culmen. The third lobule is subject to a wide range of variation, and is frequently nothing else than a small branch of the fourth lobule. In other words, the fissure (figure 4, *fiss. a*) which separates it from the rest of the culmen may be very shallow. In other cases, however, it may be as deep as (or even deeper than) the fissura praeculminata. These variations in the adult organ are due to the fact that the fissure *a* may develop before, at the same time as, or long after the fissura praeculminata. Wherefore in human embryos of the fourth and fifth months the appearance of the anterior lobe is very variable. In my own specimens the condition represented in figure 4 (*i.e.* the fissura praeculminata is the earliest and deepest) is the most usual, but the number of my specimens (four) of this period does not justify a generalisation (compare Retzius, Taf. iv. especially figure 23; Taf. ix. figure 6; Taf. x. figure 12; Taf. xxv. figures 5 and 6; Taf. lxx. figure 1; and Taf. xli. figure 1, among many others). In some mammals fissure *a* seems to be constantly more precocious than the preculminate fissure.

In the fourth month the posterior limit of the lunate area (Kölliker's lobulus lunatus posterior) becomes mapped out by a laterally-situated symmetrical pair of post-lunate fissures (sulcus superior posterior, sulcus postclivalis) [see Retzius, Taf. xxxix. figure 2; Taf. viii. figures 3, 4, 7, 8 and 11; Taf. ii. figure 7; in Kuithan's figure 21 (reproduced in Cunningham's "Text-Book of Anatomy," fig. 362, *c*, page 489), these post-lunate fissures are erroneously labelled "*sh*" (sulcus horizontalis magnus)]. According to Stroud's figures (*vide* plate vi. figures 61 and 63) the post-lunate fissures are already present in a foetus 9 cm. in

length; but, according to Kuishan, "Die Hemisphären sind noch völlig glatt" (p. 34), in a foetus 12 cm. long. In one of my own specimens, 14 cm. long, the fissures are deep and fully formed (figure 5).

If we leave out of account the floccular and parafloccular fissures, the fissura post-lunata (postclivalis) is the first furrow to be developed in the ala (or so-called lateral lobe) of the cerebellum other than by the lateral extension of the "vermian" fissures. The post-lunate fissures begin in the alæ and extend mesially;

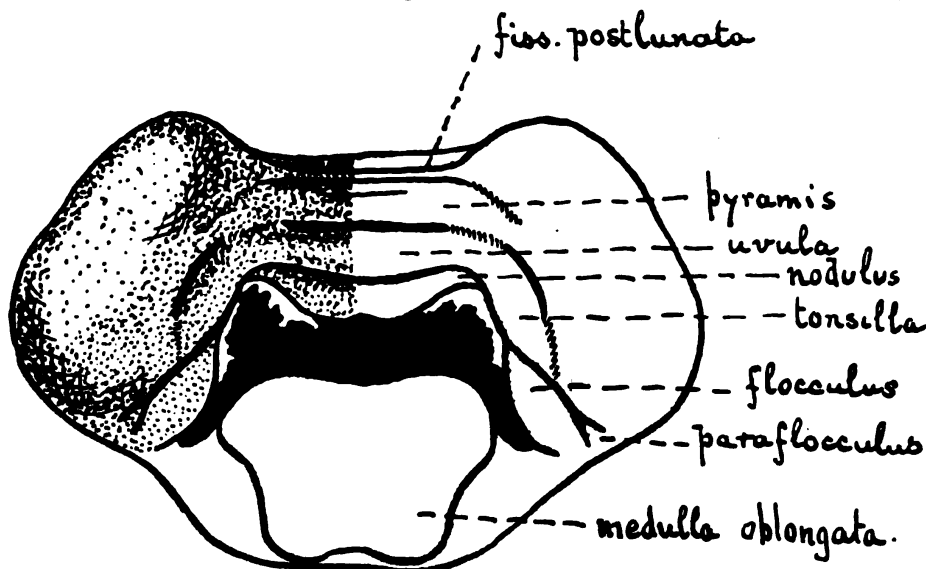


Fig. 7. The inferior (caudal) aspect of the same cerebellum represented in figure 5,  $\times 4\frac{1}{2}$ .

the two fissures often, but by no means always, become confluent on the vermis.

Most writers imagine that this fissure, by reason of its precocity, must be the "sulcus horizontalis magnus," but that morphologically unimportant furrow does not develop until much later. Stroud (5) was, I believe, the first to call attention to the fallacy of the common interpretation.

Up till the end of the fourth month the developmental history of the human cerebellum agrees in the main with that of other mammals, but from thence onward some noteworthy distinctive differences become manifest.

At the end of the fourth or the beginning of the fifth month a pair of laterally-placed post-tonsillar fissures make their appearance on the ventro-caudal aspect of the cerebellum (figure 7).

The precocious appearance of these fissures is characteristic of the human brain, as also is the great development of the prominent egg-shaped tonsilla, which is such an obtrusive feature on the lower (caudal) aspect of the organ in the latter part of the fifth month (figures 7 and 8).

In the human brain the post-tonsillar fissures usually extend mesially and become confluent with the fissura secunda.

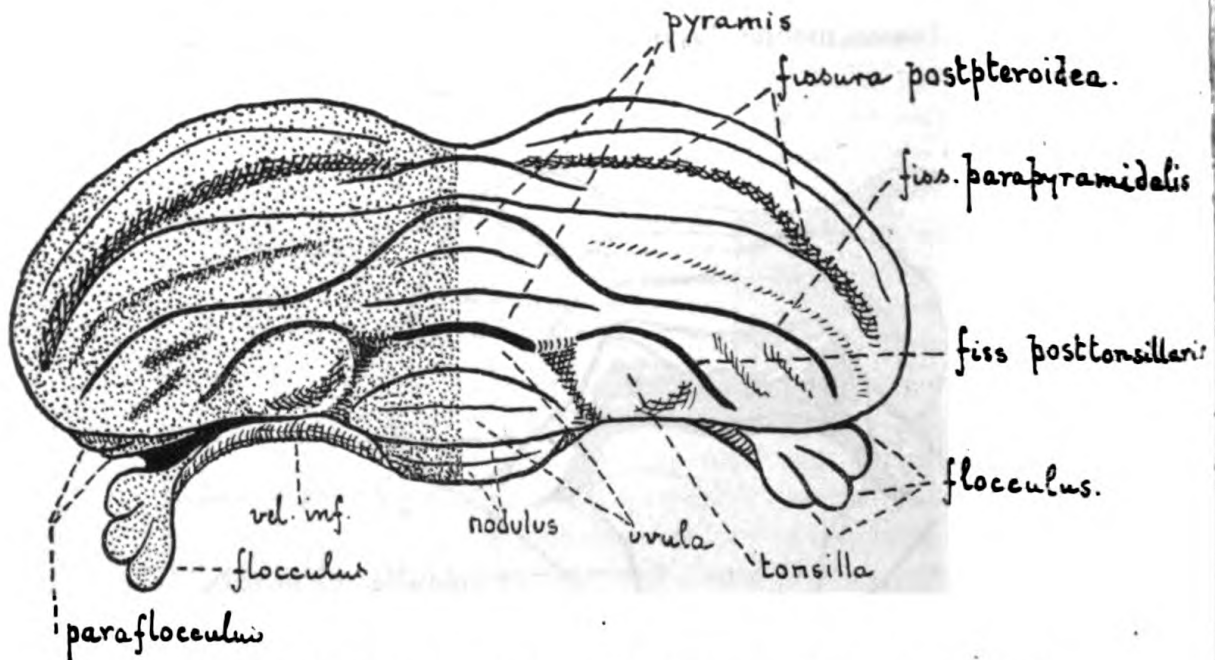


Fig. 8. The inferior (caudal) aspect of the same cerebellum represented in figure 6,  $\times 4\frac{1}{2}$ .

The left floculus has been pulled forward to expose the parafloculus and the deep fissura flocularis separating them.

The fissure between the pyramid and the uvula is the fissura secunda: that in front of the uvula is the postnodularis: that behind the pyramid is the suprapyramidal.

The result of this is that the continuity of the tonsil and the pyramid (which is found in almost every other mammal) becomes interrupted, and hence the tonsil is commonly regarded in Human Anatomy as an appendage of the uvula (posterior lobe) instead of being merely the ventral (anterior) lobule of the ala lobi medii. The direction of the folia on the uvula (figure 8) shows that the influence of the common mammalian pattern still exercises a bias on this lobule. The paravernian vallecule, which begins to develop between the uvula and tonsil—the first indication of a

distinction between vermis and ala—at the end of the fifth month, often completely cuts the connection between the two lobules; it sometimes happens that the common mammalian connection between tonsil and pyramid is revealed in the human brain by a series of submerged folia passing from the former into the fissura secunda, thence on to the lower (anterior) surface of the pyramid.

It may even happen [if I rightly interpret Stroud's figure 62 (which I reproduce here with several corrections as figure 9)]

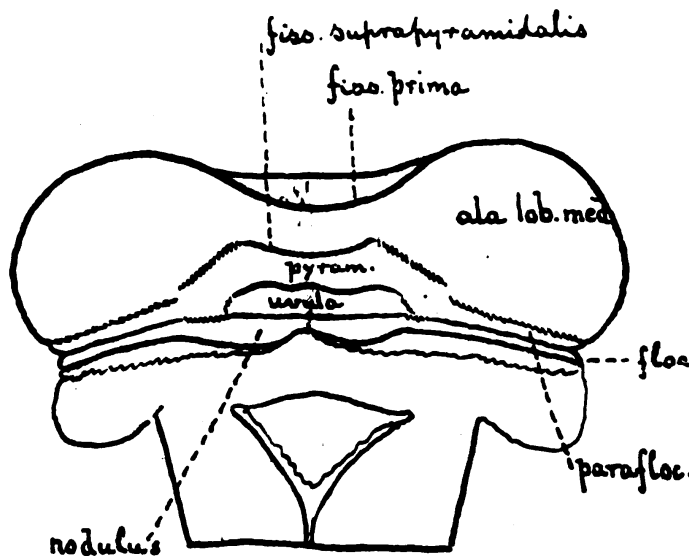


Figure 9. A diagram representing the lower (caudal) aspect of the cerebellum and the dorsal aspect of the medulla oblongata of a human embryo, 9.5 cm. long×6.7. Modified from Stroud's figure 62.

that the peculiar connection between the pyramid and the para-flocculus (copula pyramidis) which is found in many mammals, may be revealed in the early human cerebellum.

At about the middle of the fifth month a symmetrical pair of fissures make their appearance on the lower (caudal) aspect behind the post-tonsillar fissures. These parapyramidal fissures mark the posterior limit of the "biventral lobules" (figure 8); they soon extend mesially and fuse with the suprapyramidal fissure. The name parapyramidal is therefore not necessary in Human, as it is in Comparative, Anatomy, where we often have to deal with three independent furrows.

It is not until the end of the fifth or the beginning of the

sixth month that the fissura postpteroidea \* ("sulcus horizontalis magnus") makes its appearance. By this time the anterior lobe is already split up into folia (figure 6); the lunate area of the middle lobe is also subdivided by several fissures (figures 5 and 6), the flocculus, the paraflocculus, the nodulus, the uvula, the pyramid, the tonsil and the biventral lobule are all mapped out. This fissure is as late in appearing in the human brain as it is in the mammalian series. It is a secondary furrow in every sense of the term, and is morphologically of little importance. Yet this is the fissure which is regarded as of fundamental importance as a dividing line between the lobes in Human Anatomy. It begins as a depression below the anterior extremity of the post-lunate fissure (figure 5), and extends horizontally backwards; it may be formed of several furrows which are not in exactly the same plane (figure 6); this explains the frequent occurrence of large groups of submerged annectant gyri which cross the fissure in the adult brain. It gradually approaches the mesial plane and *sometimes* fuses with the corresponding fissure of the other side. It commonly happens that no such junction occurs; in the apes it is quite exceptional for such a fusion to occur. This shows how absurd is the error, committed by many writers, of identifying as the "sulcus horizontalis magnus" the deepest furrow crossing the vermis, whereas the fissure is never deep and is often absent altogether in the mesial plane. The earliest phases of the fissura postpteroidea are well shown in Retzius' illustrations, 'Taf. iii. figures 7, 8, 11, 12, 13, 16 and 17; 'Taf. x. figures 9 and 10, and 'Taf. xxxix. figures 1 and 2.

By the middle of the sixth month the cerebellum has thus been divided into its chief lobules. Its further history consists of the repeated subdivision of these lobules by means of innumerable subsidiary fissures.

The cerebellum consists of the flocculi and three chief lobes, anterior, middle and posterior.

The middle lobe alone is divisible into a mesial part or vermis and lateral lobes or alæ. The vermis (lobi medii) is divisible into a pars dorsalis and a pars ventralis (pyramid). The subdivision of the pars dorsalis into clivus, folium cacuminis and

\* In most mammalian cerebella each lateral pole is formed of a boss with a feather-like (pteron-oidea) pattern of folia. This I have called area pteroidea, and its caudal limiting furrow the fissura postpteroidea.



tuber valvulæ is meaningless and useless and might with advantage be discarded.

Each ala (lobi medii) is subdivided into a pars lunata ("lobus lunatus posterior"), a pars pteroidea ("lobus postero-superior"), a pars postpteroidea ("lobus postero-inferior"), a pars biventralis, and the tonsilla. In the lowlier Primates and in most other mammals the pars postpteroidea, pars biventralis and the tonsilla together form a narrow vertical worm-like column alongside the vermis. As it is convenient to have a name for this column I have called it the paravermis.

In man the paraflocculus consists of two or three insignificant folia attached to the lateral extremity of the tonsil with the lowermost folia of which its cortex is often joined.

The advantage of employing such a mode of subdivision of the cerebellum as I have outlined is that it permits accurate comparisons to be made with the organ in other mammals and thus opens the way for the interpretation in Man of the facts obtained by the study of the common mammalian forms of cerebellum.

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### A CONTRIBUTION TO OUR KNOWLEDGE OF THE COURSE OF THE LYMPH STREAM IN THE SPINAL ROOTS AND CORD.

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Prestwich, Lancashire.

THE following is an account of the case from which I obtained the material upon which this paper is based:—

E. S., aged 31, married, four children. The cause of her illness was assigned to domestic trouble.

She was admitted to this asylum on October 7, 1902. She was a woman of middle height, apparently in indifferent health, with an acne rash over the face. Her pupils reacted normally, her knee-jerks were lively.

She was very excited; said she had made herself ill fretting about her father and mother, who had died within the last two years; confessed to hearing "rumbling noises as of people talking in the street."

She remained depressed and fretful until October 29th, on which date she developed a boil on the posterior aspect of the right forearm. This improved, but mentally she became worse, and on November 5th one finds noted, "She is mischievous, bites her fingers and pulls her hair out." She improved a little until December 15th, when she again relapsed.

On January 16, 1903, a small abscess developed in the right axilla, with enlarged glands around. This was opened and the pus evacuated. On this day she also complained of pain in the left shoulder, which at first was thought to be due to rheumatism. Nothing unusual was observed in this region until January 23rd, when she complained of weakness and continued pain.

On examination the following condition was found present: Much atrophy of the body muscles generally, but more marked in the muscles of the left shoulder girdle. The atrophy was very distinct in the left pectoralis major, deltoid, and trapezius. She could perform all movements well, with the exception of raising the hand to the head. When she attempted this, she could only get her arm into the horizontal position; but if one placed the tips of the fingers on the side of the head for her, she was in this way able to drag the arm up until her finger tips touched the vertex.

January 24th, she appeared to be very ill, and in addition to the loss of power and wasting in the arm, there was some swelling and tenderness to pressure behind the upper part of the left sterno-mastoid. Pulse, 130; temperature, 101°.

Blood was taken from a vein, after careful sterilisation of the patient's skin and of the instrument. A small glass syringe was boiled, the skin was thoroughly washed with turpentine, ether, and chinosol, then a few drops of blood were withdrawn and allowed to run down into an agar tube and incubated. Next

day there were found typical colonies of the staphylococcus pyogenes aureus, and film preparations from these stained in the usual manner with Gram's method. During the 25th and 26th her condition continued much the same, notwithstanding the injection of anti-streptococcus serum, which was repeated at intervals until the 28th. Mentally she was very irritable and anxious.

On January 27th she was evidently worse. Pulse, 150; temperature, 102.8°. On January 28th she was more apathetic, less sensitive to pain, passed very little urine, and became, towards the evening, very drowsy. Blood was again extracted from a vein with the same aseptic precautions, and an agar tube inoculated. Death occurred at 10.15 that night. From the second agar tube staphylococci were again successfully grown.

At the autopsy the following points of interest were noted:— There were infarcts in both lungs, an abscess in the right kidney, and in the central nervous system there was unusual pallor of the grey matter. There was no lesion of the intestine. In the cervical fascia on the left side, and in the septa between the deep cervical muscles, and around the trunks of the brachial plexus, there was a moderate quantity of pus. Numerous staphylococci were found in the lung infarcts, the kidney abscess, the spleen, and, as will be seen below, in connection with the brachial plexus and some of the spinal root ganglia. A portion of the left and right motor cortex was examined by Gram's method for organisms with a negative result. It is to the effects of the organisms on the cord that I wish specially to draw attention in this paper.

Although I examined the brachial plexus first, it will be more convenient to begin here with a description of the changes found in the posterior root ganglia of the left side.

#### CHANGES IN THE POSTERIOR ROOT GANGLIA.

The following ganglia were separated from the cord and fixed in a mixture of equal parts of saturated sublimate and picric acid. Sections were stained—after combined colloidin and paraffin embedding—with toluidin blue, Held's and Gram's methods. In addition to staining the nerve cells, the two former methods also proved of value in studying the organisms found in

the tissues, and the erythrosin in Held's method was useful as a contrast stain for the fibrous tissue and vessels.

Four ganglia were taken from the left side: 7th, 6th, 5th, 4th; and two from the right side: 6th, 8th.

Proceeding from without inwards—for it will be seen that the gross changes existed, outside the ganglionic cell layers, in the pericapsular and capsular structures—one recognised, even under low magnification, large collections of deeply stained nuclei, some of which were included in vessels and some lying outside them. With the same power they were seen to occupy a position almost entirely confined to the layers of loose areolar tissue formed by the continuation of the epidural tissue into the intervertebral foramina (Photo 1). Examining first the leucocyte collections inside the vessels of the epidural tissue, these with a high power were seen to be typical thrombi blocking the veins. These thrombi were present in great number; in fact, in the 5th ganglion almost every vein was blocked to a greater or less extent by a complete or partial thrombus. The veins not thrombosed were greatly congested, and in contrast to these one found as a general rule the arterioles practically empty.

Under a higher power the thrombi presented the following details of structure:—White and red cells, fibrin threads, and scattered everywhere in this mass numerous cocci, occurring as conglomerate masses, as smaller groups, as short chains, or as single elements. Many of the nuclei of the white cells stained very indistinctly, and, judging from their irregular outline, had undergone retrograde changes due to the action of adjacent cocci, but still one could recognise cells with a round nucleus showing variations in size and others with a larger indented one.

Outside the vessels there were large collections of blood, evidently the result of hæmorrhage into the tissue layers. These blood extravasations were of varying shape and size, some being very large, elongated, irregularly shaped collections extending for a considerable distance and pushing aside or bursting through the tissue in all directions, while others again were quite small.

In these there were always to be found micrococci, singly or in groups which at times attained considerable dimensions; but the number of leucocytes varied. Where the leucocytes were plentiful one found many fibrin threads and many cocci, and occasionally examples of the ingestion of these cocci by leucocytes.

In one instance as many as ten cocci were seen inside a cell whose nucleus was small and irregular, situated at one end of the cell body, while the cocci occupied the other. The slight loss of staining reaction and diminution of size showed the commencing destruction of the latter. Only at the edges of the extravasations could phagocytosis be seen, owing to the way in which the densely packed cells and fibrin obscured the detail in the centre.

Frequently where the hæmorrhage consisted almost exclusively of red cells a considerable growth of cocci had evidently taken place forming small dense colonies with individual cocci scattered around. Photo 2 shows a good example of this abundant growth of cocci and the absence of leucocytes.

In the dense fibrous tissue which forms the capsule of the ganglion, internal to the loose areolar tissue, there were blood extravasations, the larger of which were as a rule confined to the outer layers and showed all the characteristics of those already described. But in the inner layers nearer the nerve cells the hæmorrhages were much smaller and generally consisted of a few layers of red corpuscles arranged as a narrow band pushing aside the tissue on either side. While cocci usually accompanied these hæmorrhages, yet there were many instances where they could not be seen.

In the sections of the ganglia a small portion of the nerve was included on either side, and in the areolar tissue around the latter the same changes were plainly visible as have been described. Many hæmorrhages into this tissue were seen, some of which lay close to the nerve sheath and ran along its outer aspect; but at no point did the blood or cocci pass inwards amongst the nerve fibres.

Within the capsule amidst the nerve cells the dilated capillaries and venules contained cocci either in small numbers, or in fairly large masses. These vessels were packed with red cells and in some cases with many white ones; and in two of the ganglia, viz., the 6th and 7th, there was a hæmorrhage into the connective tissue with many cocci lying in it. Although these cocci were in close proximity to the nerve cells, yet in no instance did they penetrate the pericellular capsule to attack the cells directly. In ganglia 4 and 5 the cocci were strictly confined to the vessels.

It is evident from the above that the point of greatest intensity of the pathological process was undoubtedly in the loose areolar tissue attached to the ganglion capsule. Following on an infective thrombosis hæmorrhage has taken place, the blood burrowing its way in all directions through the loose unresisting tissue, accompanied by cocci which at many points have proliferated, giving rise to colonies of considerable size. The true capsule of the ganglion, however, where the tissue is very dense and more resistant, has formed a barrier to the spread of the blood and cocci inwards.

#### NERVE CELL CHANGES.

I do not propose to enter into a detailed account of the chromatolytic change met with in the nerve cells, as this subject has now an abundant literature to which this case furnishes nothing additional. In the ganglia taken from the left side very few of the cells failed to show chromatolysis. Some of the large cells showed disintegrative changes of the central elements with a central nucleus, while in the medium-sized cells with large elements there was a central chromatolysis, but the nucleus assumed the peripheral position found in the reactive phases of this cell type. The degree of change varied of course from slight disintegration to entire removal of the chromophile granules with increasing pallor of the cell and its nucleus, and at this stage of degeneration the tendency to invasion by the proliferating capsular nuclei was very evident.

But there is a type of cell and nuclear change quite different from that just described. On examining a section stained with toluidin blue one was struck by the affinity which a great number of the cells possessed for the dye. Not only was the cytoplasm intensely coloured, but also many of the nuclei. With a high magnification one could distinguish degrees in the depth of colour of these cells, some being so deeply and diffusely stained that granules were only visible at the cell periphery or not at all. At the same time the nucleus stained homogeneously and might or might not be in the second stage of atrophy. Often, but not always, the cell edge was stained more deeply than the central parts, and as a rule there was some diminution in the size of the cell.

This condition is that now recognised as the partial or complete coagulation necrosis fully described by Marinesco in acute encephalitis and observed by others in various conditions, *e.g.* acute delirium, chorea with septicæmia, after induction of acute anæmia, etc., and I have previously drawn attention to the appearances in a paper published last year (5). There seems little doubt that such a severe cytoplasmic change is an indication either of toxic poisoning or of some other grave interference with the nerve cell nutrition, and judging from the unusually numerous examples to be found in the ganglia of this case where cocci were in close proximity to the cells and in such numbers around the ganglion, one is justified—in this instance at all events—in ascribing the necrosis to the effects of toxic influence.

The recent experimental work of Homen (4) supports this view, for after injection of staphylococci into the sciatic nerve of rabbits, although the cocci were not carried quite so far as the root ganglia, yet the nerve cells showed the early phases of coagulation necrosis as well as chromatolysis, as a result of the upward passage of the toxins in the lymph stream.

Turner (6) in a series of cases published recently has found in the posterior root ganglia of one of his cases many organisms having the appearance of the bacillus of malignant œdema and has described the cells as suffering from “acute cell change.” Unfortunately the way in which he employs the terms coagulation necrosis and pyrexial change—under the elastic heading of acute cell change—to indicate one and the same condition, leaves the reader in doubt as to the particular type of nerve cell alteration he wishes to refer to.

Pyrexial change and coagulation necrosis cannot be used as synonymous terms, as it has been an accepted fact for some time that the changes observed in experimental hyperpyrexia can easily be differentiated from those seen in coagulation necrosis. Under the heading “dark staining of cells” he gives a good description—with the exception of some confusion at the end—of coagulation necrosis as observed by previous workers, but as there seems no present need of a new term to describe the condition, one feels inclined to continue to use that originally adopted by Marinesco.

Turner’s observation of micro-organisms in the root ganglia is a very interesting one in view of the fact that the nerve cells are

markedly altered and show the above mentioned "acute cell change," which term certainly describes the condition shown in the excellent photographs accompanying the article. One is struck by the similarity the figures in his paper bear (especially 8) to the chromophile dissolution found in temperature change, and it will be seen at a glance that fig. 8—and to a less extent fig. 7—are the very antithesis of what one observes in the densely stained necrosed cells, for in this latter condition there is no chromophile dissolution—such as one finds in pyrexial change—but a dense fusing together and coagulation of the individual elements giving to the cell its characteristic hyperchromic appearance. To bracket coagulation necrosis and pyrexial change is therefore wrong when one considers that the observations on both points have been quite sufficient to clearly establish each condition as a perfectly distinct one.

#### CHANGES IN THE NERVE CELL NUCLEUS.

There was a very large amount of nuclear degeneration in cells showing chromatolysis as well as in the necrosed cells just described. Nuclei could be seen either in the first stage of homogeneity or in the secondary one of increased homogeneity with atrophy and crenation. The appearances in this case are the same as described by me in an earlier publication and still agree with the descriptions of Sarbó, De Buck and De Moor, Righetti, Schüpfer and others (5); and as my examination of this case only confirms what I observed previously, my views upon the relationship of nuclear atrophy to coagulation necrosis of the nerve cell remain unaltered, *i.e.* that the former may be the precursor of the latter. The crenation of the nucleus which one so often notices in the secondary atrophic stage could plainly be seen even with a low power, and is merely a further stage in the atrophic process. In Photo 5 one sees a typical example. Even to the end the nucleolus is visible in the deeply stained mass. The presence of so many nuclei altered in the above manner with the intimate association of cocci in the ganglia is strongly suggestive of the toxic origin of the condition.

The 6th and 8th cervical ganglia of the right side were stained and examined to compare especially the nerve cells with those on the left. Here one rarely found coagulation necrosis ;





and although a considerable amount of nuclear change was present, yet this was met with less frequently than on the left side, and usually in the first stage. There were no thrombi in the tissues around the ganglia, but here and there little hæmorrhages in the areolar tissue and rarely in the capsule. A few scattered cocci accompanied these. Amongst the nerve cell layers the vessels never showed micro-organisms.

#### CHANGES IN THE CERVICAL CORD.

As it seemed more useful to study the fibre lesions in this region the cord was hardened in bichromate of potash 2%, and thereafter treated with my own modification of Marchi's method. From the mid-cervical region two small pieces were cut out, one stained by Gram's method for organisms, the other with hæmatoxylin and eosin. It was early ascertained that there was a definite lesion present, so that one naturally preferred to concentrate one's attention on the Marchi reaction and follow out its distribution as closely as possible. Unfortunately, in extracting the cord, the posterior columns were damaged by the forceps just below the 8th cervical segment and this portion was discarded. This accident, however, does not affect the conclusions to be drawn from the presence of the lesion or prevent one from determining the inferior limit of it. As the two photographs (3 and 4) were both taken from the *6th segment*, it will be convenient to describe this one first. Commencing on the left side one found marked degeneration in the fibres of the root zone as they passed into the cord behind the horn and also of the fibres immediately outside the cord for a very short distance. The extra- and intra-medullary degenerations were sharply cut off from each other exactly at the point of entrance of the sensory fibres (Photo 3). Beyond this very small patch of Marchi reaction there was no other sign of degeneration in the piece of root attached to the cord and sectioned with it. The intra-medullary portion could be followed inwards in the form of black droplets arranged longitudinally until the fibres passed into the cuneate fasciculus. This column contained many degenerated fibres scattered especially throughout its middle part, leaving the most ventral and dorsal parts almost free. In both postero-internal columns there were fibres showing the Marchi

reaction, though not in such great numbers as in the left external column, although on either side of the median fissure one could again distinctly see a definite increase of reaction (Fig. 2). On the right side the fibres of the root zone showed exactly the same appearances as noticed on the left, but the degree of degeneration was not so great. Photo 4 is representative of the amount of change present in the right root, and when compared with Photo 3 the difference is obvious. The degenerated fibres were here again seen running into the cuneate fasciculus in which there was degeneration but not nearly so much as on the left side.

On passing round the cord from behind forwards one found that along many of the septa derived from the pia-arachnoid there were compact rows of black myelin droplets running inwards towards the grey matter, and in some cases following the course of the blood-vessels into it. Here and there in the posterior and lateral regions of the cord there were isolated black globules lying underneath the pia and separated from each other by irregular intervals.

In the anterior region on both sides there was considerable degeneration seen in the fibres of the anterior roots. Under the pia in this region there were large collections of altered myelin and rows of Marchi droplets marked out the course of the motor fibres as they passed along the septa to gain the periphery. The reaction in the septa could be followed as far inwards as the grey matter. In the roots outside the cord there was no Marchi reaction. Here and there on either side of the anterior median septum and around the finer septa given off from it there were little collections of degenerated myelin.

In both anterior and posterior horns there was abundant Marchi reaction more marked in the anterior ones and to a slightly greater degree in the left. The degeneration often showed in the form of large and fine scattered droplets, but many of these at times combined to form distinct rows. Around the cells of the left anterior horn these degenerated fibres were very prominent. There was marked degeneration of the anterior commissure. Immediately surrounding the vessels in both white and grey matter there were often to be seen large collections of black globules. Sometimes there was considerable dilatation of the perivascular spaces, but such was not always the case.

Every vessel in the meninges and in the septa showed a marked degree of congestion. In the lateral and anterior columns a few isolated degenerated fibres were found scattered irregularly throughout.

*5th Segment.*—The degeneration in the posterior roots showed just as markedly as in the 6th, and in the left and right postero-external columns occupied the same position. There were many degenerated fibres along the whole length of the median fissure on both sides and towards the commissure much degenerated myelin in the fissure itself. In all other parts of the section the degenerations were identical with those in the 6th segment with the exception of a slight increase in the amount of altered myelin seen in the anterior cornua of both sides, which was still more marked on the left. Further, on the left side in the mesial aspect of the grey matter at its base there was a small hæmorrhage in whose midst lay globules of altered myelin. The vessels again were markedly congested with degeneration around, and in the septa there was no decrease in the amount of Marchi reaction. As in the 6th segment the great bulk of the degeneration was in the left cuneate fasciculus.

*4th Segment.*—In the intra-medullary portion of the sensory roots and in the extra-medullary for a very short distance there was degeneration; much less, however, than that found in the 5th and 6th segments and equal on both sides. On the left side this degeneration was continued into the postero-external column and was most abundant where the fibres entered the column. As one passed ventrally the degenerated fibres rapidly decreased in numbers, and, as in the case of the roots, the total amount of degeneration was much less than that in the preceding segments. From this column there were to be seen a few bundles of collaterals running into the posterior grey horns, and showing the Marchi reaction. In both postero-internal columns there were a few scattered black droplets, and in the right postero-external one the degeneration was very slight.

In this segment there was still almost as much degeneration in the anterior grey matter, in the anterior commissure, at various places around the cord underneath the pia and in all the septa, as in the segments above described. The vessels were congested, while degenerated myelin often lay in the perivascular spaces, which were sometimes dilated. In the left anterior grey horn,

in its anterior part, a vessel had ruptured leading to a moderate degree of blood extravasation.

*3rd Segment.*—There was very little degeneration of the motor fibres in the septa, but in the posterior columns there was more than in the preceding segment, but rather less than in the 5th and 6th. The left cuneate fasciculus still showed more degeneration than the other fasciculi, but the difference was much less marked than in the preceding segments. In the two postero-internal columns the degeneration was confined to the anterior parts, and as one passed over to the right posterior horn the number of altered fibres markedly decreased. The degeneration in the left cuneate fasciculus differed from that in the other columns in its further extension backwards and also forwards, thus affecting many of the fibres just behind the commissure. In the anterior cornua the amount of degeneration was distinctly less than in segments 5 and 6. The vessels were again congested and around them one found large collections of altered myelin.

*2nd Segment.*—At the lowest part of this segment one found a marked decrease in the degeneration both in the posterior roots and in the columns. In the former the Marchi method only showed some scattered globules contrasting very markedly with the mass of degeneration found in segments 5 and 6. The left cuneate fasciculus was that most affected, and the form the degeneration assumed was roughly comma-shaped, with the head just internal to the anterior part of the posterior horn, and the tail passing down the centre of the column and curving outwards towards the root zone. The degeneration was very sparse, and more prominent anteriorly. Elsewhere in the posterior columns morbid fibres were very few in number, and had no special distribution. In the anterior commissure there was some degeneration, and this could often be seen to follow the course of a vessel passing in from the anterior median septum. The septa, anterior grey horns, and the white matter immediately underneath the pia still showed Marchi reaction, but rather less in degree than in the lower segments.

In sections taken from the highest part of the segment the degeneration had practically gone. There was nothing of it in the roots on either side, and in the left cuneate fasciculus all that remained of the comma-shaped degeneration was simply a

few fibres retaining somewhat the same distribution as in the lower part of the segment, but very much less marked. In the remainder of the posterior region there was nothing abnormal with the exception of a small group of fine Marchi droplets on either side of the postero-median septum, situated somewhat nearer the dorsal than the ventral surface of the cord. The grey matter on the right side was clear, but in the anterior commissure and in the left anterior horns there was a very slight degeneration confined to the immediate vicinity of the vessels. In a few of the septa of the lateral region of the cord there could be seen very small black myelin collections. At one or two spots around the periphery of the cord there were a few degenerated myelin globules underneath the pia. Everywhere the vessels were congested.

In order to see the inferior limit of the lesion we must go back to the point from which we started, and compare the 6th segment with those beneath it.

*7th Segment.*—There was nearly quite as much Marchi reaction here as in the 6th, and the distribution was the same, with slight differences here and there throughout the section. The preponderance of degeneration in the left root and postero-external column seen in the description of the 6th segment was also noted here, and the degeneration in the anterior grey cornua was quite as marked. In the posterior columns, just behind the commissure, there were numerous black globules, thus differing from the segment above, in which this region was almost free from degeneration. Around the vessels there was much degeneration, and in addition four small hæmorrhages in the right postero-external column in its dorsal part.

*8th Segment.*—In this segment there was a very noticeable diminution in the amount of change in every part of the section. In the posterior roots there were still a few black droplets, the left postero-external column still showed somewhat more degeneration than the corresponding one on the right side, and on either side of the median septum the internal columns showed quite as many black dots as the left external one. The septa, anterior commissure, and grey matter contained degenerated myelin, but to a much less extent than in the segment above.

As previously mentioned, the 1st dorsal segment was injured

at the autopsy, and therefore was excluded from the record of the case.

*2nd Dorsal.*—Marchi's method showed practically nothing. In the lateral columns of the cord there were a very few scattered degenerated fibres and also slight degeneration in some of the septa. In the anterior grey horns there was nothing unusual except a few black dots around three of the vessels. In the posterior columns there were a very few degenerated fibres around the postero-median septum and also in the left cuneate fasciculus. All the vessels were congested, and in the left crossed pyramidal tract there were several minute hæmorrhages.

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By Gram's method only one small group, consisting of seven cocci lying in the pia of the posterior region of the cord, was seen.

The sections stained by hæmotoxylin and eosin showed no trace of cell emigration around the vessels, or other pathological change.

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*Posterior Nerve Roots.*—On the left side, the 7th, 6th and 4th sensory and motor roots were treated by Marchi's method, and cut in transverse section, both outside and inside the dura.

In addition to this, the 6th and 7th sensory roots were teased between the dura and the cord, after having been the usual time in Marchi's fluid. On the right side, the 4th, 5th, 6th and 7th sensory and motor nerves were treated in the same way, and cut in transverse section from the level of the ganglion to the cord. There was practically no reaction to be seen. All that one found consisted in a very few isolated fibres showing the osmic acid reaction. In all the sections the vessels were greatly congested.

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*Brachial Plexus.*—The three primary cords of the brachial plexus were examined by Marchi's method for degeneration and by Gram's for organisms. By the former method only a very few black dots were seen, so that one can say that the result was negative.

By Gram's method pathological changes were found, somewhat similar to those noted in the left root ganglia, but of much

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less intensity. In the veins of the loose areolar tissue surrounding the perineurium there were thrombi containing cocci, and as a rule fewer white cells than those around the ganglia. These cocci were present in masses, few in some bundles and numerous in others, but never attaining anything like the quantity found around the ganglia. Hæmorrhages into the epineurium were rare, and those observed contained no cocci. Here and there along the septa of the nerve bundles and on the inner aspect of the perineurium a very slight degree of small cell emigration was visible, situated around the vessels.

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The first point to be borne in mind in considering this case is the fact that on two separate occasions the *staphylococcus pyogenes aureus* was cultivated from the blood—under the strictest aseptic precautions—so that, although one's attention is mainly directed to the lesions in the cervical region, yet the infection of the blood stream must not be lost sight of, especially when one comes to discuss the origin of the degeneration in the cord.

How the patient became infected is a matter rather difficult to determine, but, as we have seen from the clinical history that she was in the habit of pulling out her hair, and that she developed abscesses in the course of her illness, then the skin may be looked upon as the probable source.

The local lesion in the cervical cord and ganglia is of the greatest interest in connection with the lymphatic system in these regions, as the changes can be brought into line with the experimental work which has recently been done on the course of the lymph stream in the peripheral nerves and cord. From the details given above, one cannot fail to be struck by the fact that the bacteria were concentrated around the left spinal root ganglia already mentioned, and had spread very slightly upwards, for none were found in the dura or in the loose tissue covering it, nor in the cord substance; and although one did find a small group of staphylococci in the pia, yet this growth was so limited as to justify one in leaving it out of account. The nerve on the central side of the ganglion undoubtedly showed bacteria and hæmorrhages for a short distance, and I admit that one would have done well to have determined the highest limit of

the bacterial spread with accuracy, but when the distribution of the Marchi reaction in the cord was observed I treated the roots with osmic acid, in the firm belief that degeneration would be found in their whole length. As we have seen, this preconceived idea was quite an erroneous one, the result of examination being negative.

In the cord, then, we have to deal with a lesion not due to the direct action of bacteria upon the fibres, but to the upward passage of toxins in the lymph stream derived from organisms situated peripherally. Without the aid of the excellent work of D'Abundo (1), Guillain (2), and of Homen (4), on the course of the lymph stream in the peripheral nerves and cord, one would have been greatly handicapped in trying to explain the cause and distribution of the lesion in the cord.

The course which the lymph takes in the cord and nerves has been studied on two lines, viz., by the injection of China ink, and by the injection of bacteria; and the direction in which these spread has been followed in sections made above and below the point of injection.

D'Abundo (1) has shown that if China ink be injected into the posterior columns of a dog the spread of the granules is in a definite direction. In the first place, should the posterior columns be alone affected, then the posterior roots act as barriers to the spread of the granules laterally. These pass for the most part upwards from the point of injection, gradually diminishing in numbers from below upwards, and sometimes can be detected in the central grey matter. After injection into the central grey substance the granules affect the posterior columns, grey matter, the lateral columns slightly, and spread in the direction of the anterior pyramidal tracts. There is a gradual decrease in number from below upwards, until 15 mm. above the injection point, they are limited to the posterior column and dilated central canal. Higher up still they may be limited to a small central oval patch in the posterior columns. Below the point of injection they are rapidly limited in distribution and finally are seen in the perivascular lymph sheaths. Guillain (2) conducted his experiments on similar lines, and found that after injection of China ink into the posterior columns the granules passed upwards and at the same time showed a tendency to travel towards the central canal and even to pass into it. Further, they



were noted in the adventitial sheath of the artery in the anterior median fissure, and slightly also in the antero-lateral columns. The author's principal conclusions are, that the circulation of the lymph in the cord is ascending, that the circulation of lymph in the posterior columns is independent of that in the antero-lateral ones, and that the central canal is a lymphatic channel.

In a more recent paper written in conjunction with Marie (3), he adds that the lymphatic system of the posterior meninges communicates very little or not at all with that of the antero-lateral meninges, and that there is a special system for the posterior columns and adjacent pia. Guillain has found that the diffusion of coloured fluids can also occur when the injections are made in the region between the pia and cord.

Homen's work (4) on the action of staphylococci on the peripheral nerves, spinal ganglia and cord, explains why one failed to find cocci in the dura or cord when so many existed around the ganglia of this case. After injecting staphylococci of increased virulence into the centre of the sciatic nerve, he found that the tendency for the organism to spread upwards was very slight, and that the cocci never reached the ganglia or the cord. A few cm. central to the point of injection—at the end of twenty-four hours—the cocci were found principally in and about the lymph spaces on the inner side of the perineurium, lying in and between numerous leucocytes. He further found that after a few days the cocci tended to disappear, until on the ninth or tenth day they could be seen undergoing degeneration.

Although in Homen's experiments the cocci were introduced into the centre of the nerve, while in my case they were separated from the nervous structures by the sheath, and therefore the local effects produced in the two cases were quite different, yet his observation that the cocci possessed little power of spreading upwards is of great value, as the spinal ganglion cells and nuclei in his experiments showed changes, evidently the result of toxins exerting their influence at some distance central to the seat of the bacteria, a fact strongly in favour of the theory that in the nerve the flow of lymph is upwards. This view is supported by Homen's further experiments. If other organisms are injected, such as the pneumococcus or streptococcus, he has found that these can spread up to the root ganglia and to the cord, affecting especially the membranes of

the latter. In the ganglion the changes were found chiefly in the capsule and the neighbouring nervous elements. There were changes in the roots, small in degree, which occurred sooner in the posterior than in the anterior ones. In the spinal meninges there was a small-celled infiltration which passed into the cord along the septa, and here he found alteration of the adjoining fibres.

Homen also injected staphylococci directly into the cord, death following in a few days, and found that the spread of the cocci took place especially in the meninges and in the central canal. In the latter the diffusion occurred more rapidly, and the cocci were seen to pass out between the lining cells into the surrounding grey substance. The changes induced were practically limited to the point of injection and its surroundings, congestion of the vessels and hæmorrhages being amongst the effects observed. In the root ganglia and sciatic nerves examination for bacteria or their effects proved negative. With the injection of the filtrate of a twenty-four hours old bouillon culture the author found slight changes in the nerve amongst which were hæmorrhages. As one passed centrally all the changes rapidly decreased.

Possessing a knowledge of the facts elicited by the above workers, one has little difficulty in interpreting the pathological changes found in the cord of this case and in tracing their direct association with the toxins from the bacterial growth around the ganglia.

It is probable that the infective process started in the cervical fascia and in the septa amongst the deep cervical muscles, and that from this situation the organisms passed into the intervertebral foramina infecting the loose areolar tissue there and giving rise to the changes which we have seen. From the large coccal growth around the capsule and in it, there must have been a steady upward flow of toxic lymph in the channels of the nerve sheath, this structure owing to its density protecting the nerves from injury until the point of entrance into the cord was reached. The toxins must have travelled more quickly along the sensory nerves, for it was in the intramedullary part of this system that the lesion was most marked. On reaching the cord by the posterior roots, the toxins were apparently compelled to take two paths, one into the posterior columns, and the other into

the pia-arachnoid with which the nerve sheath is continuous. The degenerations traced by Marchi's method now show us the further course of the toxic lymph and also the points at which its influence was exerted most.

Judging from the degree of degeneration in the left sensory root zone and cuneate fasciculus, the toxins must have passed in large quantity into the left posterior column, and in Photo 3 one sees that the degeneration has commenced immediately at the point of entrance of the sensory fibres into that column. It is a significant fact that just at the point of entrance into the cord the incoming fibres lose their neurilemma, and thus are left exposed to the full influence of poisons in their immediate neighbourhood. As a matter of fact, they were bathed in the toxins flowing into the cord at this point. In the extra-medullary part of the root there was degeneration for a very short distance backwards, which no doubt was toxic in origin also.

The degeneration of the intra-medullary part of the motor nerves can be similarly explained by the upward passage of toxins in their sheaths and the fact that they show less degeneration than the sensory nerves, is in agreement with Homen's view that toxins are carried up in the posterior roots more readily than in the motor. The lymph coming up the motor nerve sheaths must have taken two directions, the main stream passing down the septa towards the grey matter and attacking the fibres in its course, while a smaller stream travelled along the meninges towards the antero-lateral parts of the cord. In the meninges, the path taken by the toxin-laden lymph can be traced around the cord on both sides, and in the posterior region its course is indicated by the degeneration in and around the postero-median fissure, and further probably by the degeneration in the sensory fibres of the right side. Here the degeneration was rather less than that of the left side, and it is possible that the toxic lymph crossed the posterior segment of the cord; but as organisms were present in the right sixth and eighth ganglia, one has to reckon with the additional fact that toxins travelled up the right sensory and motor roots.

In the lateral region of the cord, the degeneration in the septa and underneath the pia in all probability owed its origin to the lymph coming in by both anterior and posterior roots,

passing by the meningeal spaces to all parts of the periphery of the cord and down the septa, so that the whole circumference of the cord must have been encircled by the smaller quantity of the lymph which did not enter the cord substance. At the same time one must bear in mind the fact that with the infection of the general circulation, toxins could pass out from the congested vessels in the pia and septa and attack the adjacent fibres. The hæmorrhages which were noted in the white and grey matter are proof of the toxicity of the blood. Regarding the degeneration in the grey matter, there is every probability that this owed its origin principally to the transudation of lymph from the vessels, intimately associated with which there was much degenerated myelin; but as there was slightly more of it on the left side, there seems sufficient reason for thinking that toxins had passed down the anterior septa in which the motor nerves run into the anterior part of the grey matter, while at the same time a certain amount of diffusion took place from the left posterior column into the central grey substance.

In the anterior commissure the degeneration was associated with the presence of a large vessel passing in from the anterior median fissure, so that its origin was probably partly vascular and partly due to toxins spreading out from the central canal.

#### CONCLUSIONS.

1. Although bacteria have little tendency to spread along the nerves, their toxins can be carried in the lymph stream and exert their influence some distance from their source of origin.

2. In the spinal roots the flow of lymph is upwards towards the cord.

3. Of the lymph flowing up the posterior roots, the greater part passes into the posterior columns, while a small quantity flows into the lymphatic spaces of the pia-arachnoid covering the posterior and lateral regions of the cord.

4. Where the fibres enter the cord, at which point their sheath and neurilemma are lost, they are specially vulnerable to the influence of toxins in the lymphatic system of the roots and meninges. This applies to both sensory and motor nerves. The last two conclusions are important in view of the recent opinion expressed by Marie and Guillain (3) on the etiology of tabes,

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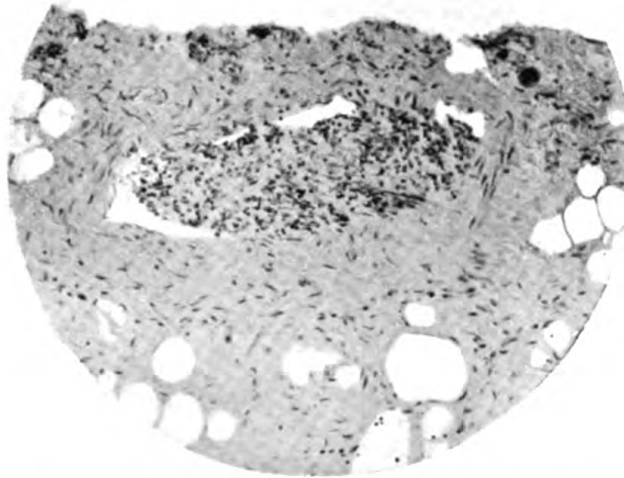


PHOTO 1. Thrombosed vein in the loose areolar tissue around the capsule of the ganglion. Held's method.

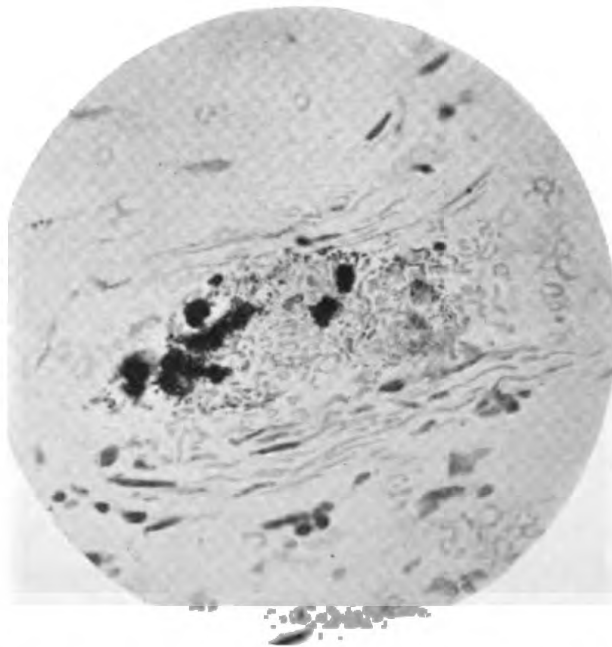


PHOTO 2. Groups of staphylococci in a hæmorrhage into the ganglion capsule. Observe the absence of leucocytes. Tolindin blue.



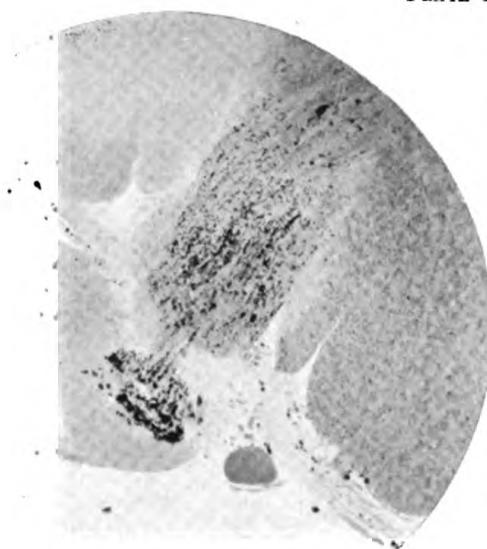


PHOTO 3. Sixth cervical segment; left side. Note the degeneration of the sensory fibres. Marchi's method.

PHOTO 4. Sixth cervical segment; right side. Compare the degeneration of the sensory fibres with those of the left side. Marchi's method.

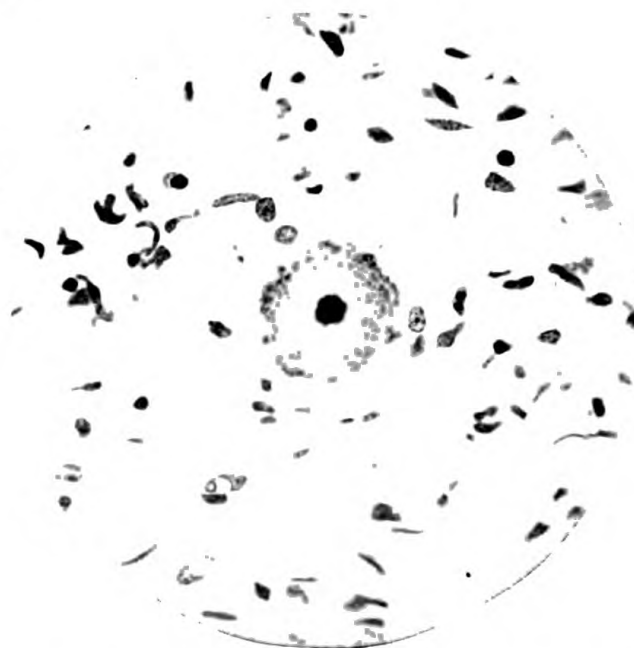
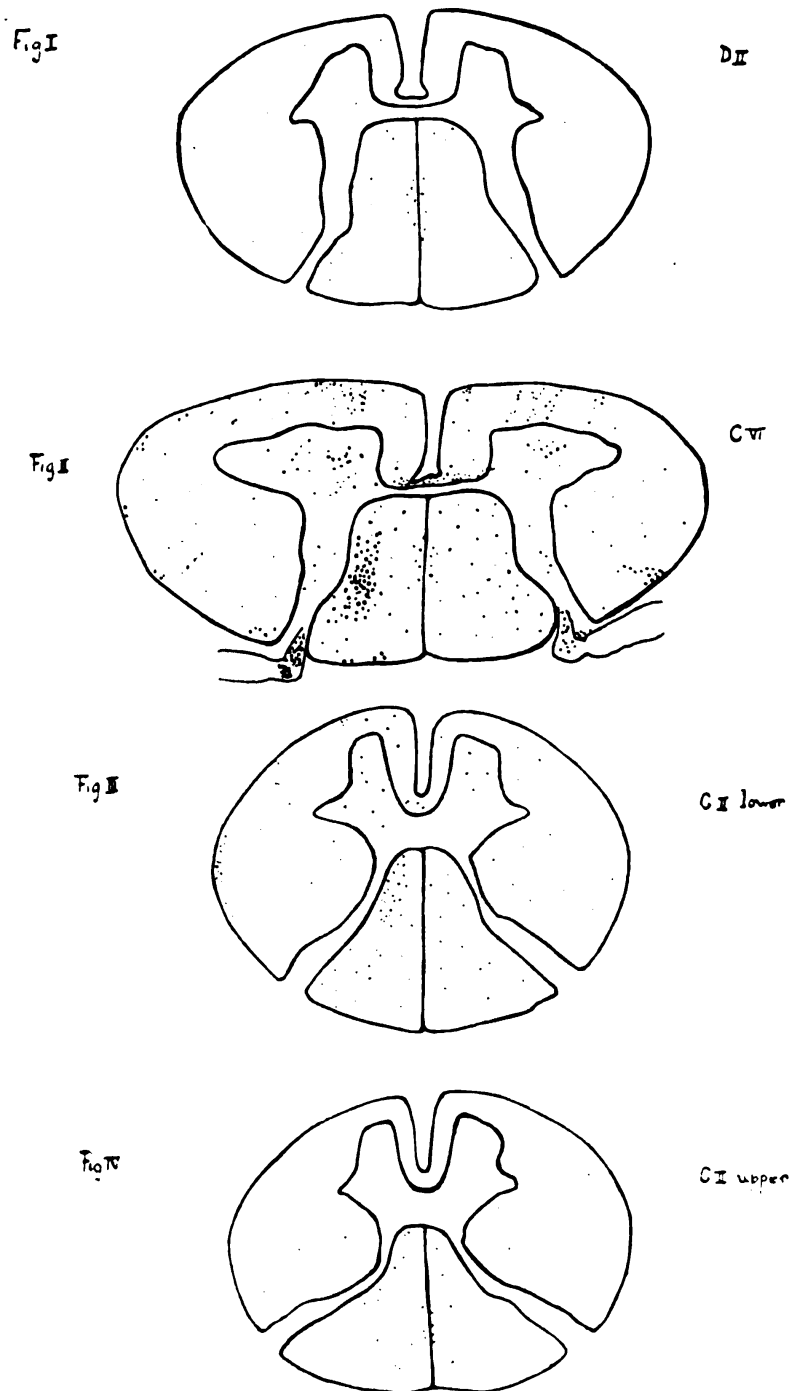


PHOTO 5. A degenerated posterior root ganglion cell, with the nucleus homogeneously stained, atrophied, and crenated. Toluidin blue.







These drawings are intended to show the distribution of the degenerations in the cord. Note how the left cuneate fasciculus is the tract most affected in the sixth cervical segment, and contrast with C<sup>2</sup> and D<sup>2</sup>. The figures are purely diagrammatic.



viz., that this disease is due to a lesion—probably syphilitic—of the lymphatic system constituted by the posterior roots, pia and cord.

5. The course of the lymph stream in the posterior columns is ascending.

6. The lymphatic system of the posterior columns does not communicate with that of the lateral columns.

I am indebted to two of my colleagues for their kindness in helping me with portions of this paper, and for many valuable suggestions. Dr Scholefield carefully examined the sections of cord prepared by Gram's method and checked my observations, while Dr Lefanu is responsible for the four drawings showing the distribution of the Marchi reaction.

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### CAVITIES IN THE CORD.

By R. G ROWS, M.D.

THE subject of Cavities in the Cord has attracted a large amount of attention during the past few years, and as a result of this we find that the number and variety of morbid conditions which may lead to the production of these cavities has considerably increased. In place of the idea that all cavities in the cord are due to some developmental defect in the differentiation of certain embryonal cells, which later in life start into activity and produce an overgrowth of tissue in which degenerative changes, absorption and formation of cavities take place, we see an

extensive list of causes in Schlesinger's monograph (1) published last year.

In this list we find :—

1. Cyst formation after injuries, whether they produce hæmorrhages or not.

2. Inflammatory and non-inflammatory softening with subsequent sclerosis.

3. Syringomyelia. (a) True hydromyelia. (b) True tumour with cavities. (c) Syringomyelia gliosa. (d) Syringomyelia following vascular change without overgrowth of neuroglia. (e) Pachy- and lepto-meningitis with cavities.

Even as to the character of some of these processes authorities are not yet agreed, and especially is this the case in syringomyelia gliosa.

Hauser (2), in his "Etudes sur la Syringomyélie," suggests that instead of describing syringomyelia gliosa as a primary overgrowth of the neuroglia, even when it is associated with hydromyelia, it should be considered as an inflammatory reaction of the neuroglia to some morbid agent.

He quotes in connection with this point Turner and MacIntosh, and Phileppe and Obesthür, who have found a gliomatous appearance and an inflammatory condition together in a cord in which cavities were seen. They expressed the opinion that the difference between the two conditions is simply one of degree, and that both are the result of vascular degeneration.

Ziegler (3) states that no sharp line can be drawn between glioma, neuroglioma and diffuse sclerosis. Weigert (4) also says that the cells in the overgrowth are too few, and the fibres are too numerous, to justify the use of the term "Glioma" in the cases.

Thomas and Hauser (5) have recently published a paper in which they described cavities in the cord which, they considered, were due to an atrophy following disease of the vessels and consequent malnutrition. Many of these cavities were seen around vessels, and there was a certain amount of neuroglial overgrowth surrounding them, which produced a sclerotic condition.

They spoke of this overgrowth of the neuroglia as a reaction secondary to the disease of the vessels and the atrophy of the nervous tissues.

They quote Müller and Medin, Weitz and Marinesco as having described similar cases.

It will be seen from the above that the question of the pathogenesis of cavities in the cord is by no means yet settled.

Having in the last two years found cavities in two cords, and the cause in each case having been somewhat unusual, I have thought it might be of some interest to give a description of the conditions found, and to draw attention to some of the anatomical findings. For purposes of comparison I shall make use of a case of syringomyelia gliosa which Drs Orr and Cowen have kindly placed at my disposal.

The first case was found in a demented, bed-ridden, general paralytic, in whose cord a most remarkable condition of cavities was found.

This condition was much more marked in the upper dorsal region than in the other portions of the cord, but it was the same in kind throughout. In the dorsal region (figure 1) the grey matter on both sides of the cord was broken up in most of its extent. There was a large cavity in each posterior horn, and in the central and lateral portions of the grey matter cavities of various sizes were seen, and the nervous and neuroglial structures had atrophied to a considerable extent.

This is well seen in figures 2, 3 and 4, under increased magnification.

In figure 2 two conditions may be noticed. Laterally there is a loss of substance due to atrophy of all structures. The nerve fibres have almost entirely disappeared, and there remains only a remnant of the neuroglial network. This is also well seen in figure 4 in the tissues surrounding the central canal.

The other condition found in figure 2 consists of rounded, smooth-walled cavities of various sizes, in which a vessel is seen attached to the wall on one side. In longitudinal sections the vessel can be followed across the cavity, or along one side of it.

The walls of these cavities were formed chiefly of neuroglial fibres which had been compressed as the cavity had increased in size, and here and there a darkly stained neuroglial nucleus was found. Nerve fibres were seen in close proximity to this wall in some cases. Nowhere was there any sign of an epithelial lining.

In figure 3 the pressing together of the neuroglial fibres is better seen, and distinct layers can be distinguished.

Scattered amongst the atrophied neuroglial network were a

few irregularly shaped, darkly stained, neuroglial nuclei, but no protoplasm could be distinguished around them.

Nowhere in the cord was there any appearance which suggested an overgrowth of the neuroglial tissue.

The central canal was filled with nucleated cells, but was not larger than it often is in cases of chronic diseases of the spinal cord (tabes, multiple sclerosis, etc.) (Fig. 4).

Nowhere did these nucleated cells extend beyond the neighbourhood of the canal.

In the white matter numerous elongated clefts were seen, and in these a vessel was almost invariably found attached to one side.

Similar changes were found in the cervical and lumbar regions, but they had not reached such an advanced degree.

The condition of the vessels in this case was very striking, and was of great importance. Throughout the cord there was marked hyaline degeneration of the vessel walls (Figs. 5 and 6).

In some, muscular fibres could still be seen inside a degenerated perivascular sheath, but in many, no definite structure could be distinguished outside the intima, and the wall consisted of a homogeneous, hyaline-degenerated band. This change was found in vessels of all sizes, and was quite as advanced in the veins as in the arteries (Fig. 6). In many instances the vessel was entirely occluded.

Nowhere was there any sign of hæmorrhage into the tissues, or of small-celled infiltration into the vessel walls.

The membranes around the cord were considerably thickened.

The second case showed the condition to which the term syringomyelia gliosa is usually applied, and exhibited a marked contrast to that described above.

In this cord there was in one portion a dilated central canal, which was lined with cubical ependymal epithelium (Fig. 7). This was seen in small heaps in some parts of the wall. Around this there was an enormous overgrowth of the neuroglial tissue, cells and fibres, and small vessels were very numerous. These neuroglial nuclei were especially numerous at the edge of the growth. The nuclei were large, round, and deeply stained, and lay in a close meshwork of fibres.

In this overgrowth the processes of homogeneisation and

rarefaction, described by Schlesinger, were progressing rapidly, so that areas were seen in which the nuclei had disappeared, and a few neuroglial fibres, irregularly arranged, alone remained; in other areas these had disappeared, and the formation of cavities was going on (Fig. 9).

In another section of this cord there was a huge cavity which extended throughout most of the grey matter on both sides of the cord (Fig. 8). The wall of this cavity was formed of neuroglial overgrowth of varying thickness, and was not lined with cubical cells. The central canal, filled with ependymal cells, was still present in the anterior wall.

In still another section some portions of the wall of the cavity were lined with cubical epithelium and others not, and it was evident that in one part of the cord there was a condition of hydromyelia, and in another part a condition of syringomyelia gliosa, and that in the intermediate part these two cavities became united, owing to the atrophy and absorption of the tissue which at one time separated them. The vessels in this case also showed an advanced degree of hyaline degeneration, and the vessel wall was often represented by a band of hyaline substance outside the intima, which showed no definite structure either of muscle fibre or connective tissue.

Figure 9 shows a portion of the wall with higher magnification, and exhibits the processes of rarefaction in one part.

The third series of sections was obtained from a case of acute insanity, which ran its course in about three weeks.

In this case micrococci were found in the posterior root ganglia, and a few organisms were found in the cord itself.

Figure 10 shows a large cavity in the mid-portion of the grey matter, which had been caused by a hæmorrhage into the nervous substance, and the tissues around had been ploughed up by the same hæmorrhage. A similar cavity was present in the corresponding portion of the grey matter in the opposite side of the cord.

In this region all the vessels were acutely engorged, and in one section I was able to see a ruptured vessel lying in a cavity, and surrounded by tissue which had been broken down by the hæmorrhage.

These hæmorrhages cannot have occurred long before the

death of the patient, because the blood corpuscles were unaltered, and no trace of blood pigment was seen anywhere.

Besides these cavities produced by the hæmorrhages, there were others of quite a different character, as will be seen by looking at figure 11, which have a marked resemblance to some of those seen in the first case.

These cavities had been formed around the engorged vessels, which had remained attached to the wall of the cavity at one point.

The wall of the cavity consisted of the nervous and neuroglial tissues, which had been detached from the vessel, and pushed back by the inflammatory exudation.

In this case, also, there was an entire absence of overgrowth of the neuroglia.

It will be seen at once from the above description that in these cases we are dealing with three distinct pathological conditions.

The first was an example of what Schlesinger (1) has described as "Syringomyelia following disease of the vessels without gliosis." But this is not a full statement of the case. The vascular degeneration may have been the primary cause, but it led to a further important result, which, in its turn, augmented the morbid condition.

It is interesting to note that this vascular degeneration affected the veins almost as much as it did the arteries. In connection with this, I may refer to the article on "Tabes," by Nageotte (6), in which he states that in this disease the veins are frequently more affected than the arteries. This has been confirmed by Pansini (7), who found it to be the case in the cord of a man who had had syphilis.

In my own case the walls of many of the vessels consisted of a thick band of hyaline-degenerated tissue outside the intima, and in many instances this thickening had occurred irregularly, and projected into the lumen of the vessel, so as almost to occlude it (Fig. 5).

Now, not only did this offer a great impediment to the flow of blood along the vessels, but it destroyed the principal lymph channels in the cord, viz., those of the perivascular sheaths of the arteries and veins. The importance of this will be the more appreciated when we remember that there are no lymphatic vessels with true walls in the central nervous system.



Many observers have drawn attention to this fact. D'Abundo (8), in his work on the paths of the lymph flow in the cord, came to this conclusion; and Marie and Guillain (9), in their article on the causation of Tabes, say that "there are no walled lymphatics in the cord, as in other organs, but there exist perivascular sheaths and spaces, through which the nutritive fluids circulate." Ford Robertson (10) has maintained the same fact in connection with the flow of lymph in the brain. Lustig (11) also refers to it.

This being so, and the three factors on which the production of oedema depends, viz., the difference of pressure in the fluids within and outside of the vessels, the altered chemical composition of the fluid in the vessels, and the altered permeability of the vessel walls, being present, I think it is easy to see how these cavities around the vessels were produced. The exudation first dilated the lymph spaces, and the onflow of the lymph being impossible because of the hyaline degeneration of the perivascular sheaths, the structures around the vessels were pushed aside, and these cavities were formed.

But their formation was assisted by another factor, which has been mentioned by Ziegler, Schlesinger and others. Ziegler (3) says that "atrophy of the neuroglia is a frequent consequence of vascular degeneration," and Schlesinger says that "stagnant lymph acts as a solvent on the neuroglia."

The cord in this case must have been lymph-logged, and this produced the atrophy of the neuroglia and nerve elements which have been described, and also assisted materially in the production of the cavities.

It will be interesting here to refer to the production of the cavities around the vessels in Case III., figure 11. In this case, we find an engorged condition of the vessels following a toxic infection, and an appearance of spaces very similar to that seen in Case I.

Now in this case also, the three chief factors concerned in the production of oedema were present, and to them was added the vaso-motor paralysis, which is produced by the action of toxins on the vessel walls. Hence there was an outpouring of lymph, an oedema, which produced a dilatation of the lymph spaces, and the formation of cavities around the vessels.

In these two cases, then, the one a chronic vascular degenera-

tion, and the other an acute vascular disturbance of toxic origin, we find an obstruction to the flow of lymph, a formation of cavities, and a consequent malnutrition of the tissues of the cord.

We must also note the absence of any neuroglial proliferation in this acute case as well as in the chronic case.

Orr (12), in a case in which he found staphylococci in the blood, and also found the sheath of the posterior root ganglia crowded with the same organisms, has observed that there was no sign of proliferation of the neuroglia in the spinal cord.

Hauser (2) has described two lesions of the neuroglial tissue in his cases of syringomyelia, the one irritative, and the other destructive.

The former, he says, follows the action of some morbid agent, which stimulates the neuroglia to proliferate; whilst the latter is seen in cases in which the nutrition of the tissue is disturbed by vascular disease. Ford Robertson (10) says that proliferation of the neuroglia follows the application of an irritant of suitable kind and intensity.

Goodall (13) states that signs of proliferation may be observed within twenty-four hours of the application of the irritant. In these acute cases there can be no doubt that the irritant had acted for a much longer period than twenty-four hours, and still there was no sign of neuroglial proliferation.

In these cases I think we must assume that the irritant was of such a kind or intensity, or both combined, that the power of reaction of the neuroglia to the irritant was paralysed; whilst in the chronic case, the nutrition of the tissue was so disturbed by the vascular disease that it was impossible for it to proliferate.

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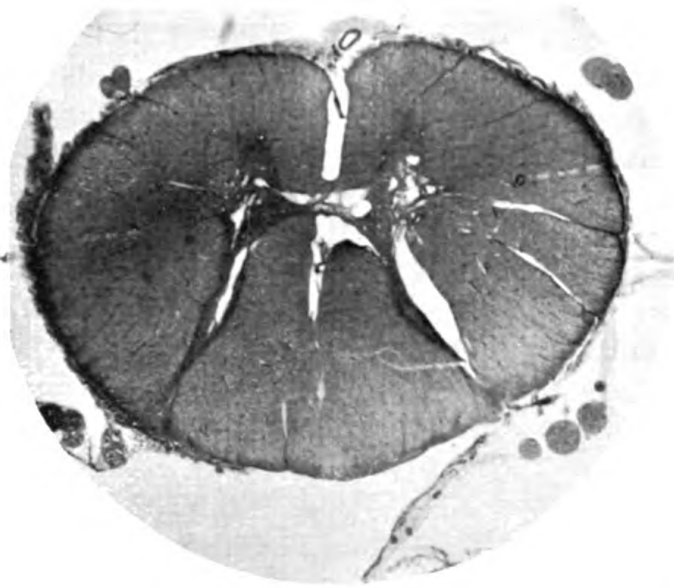


FIG. 1. Section of dorsal region of cord, showing cavities in central region.

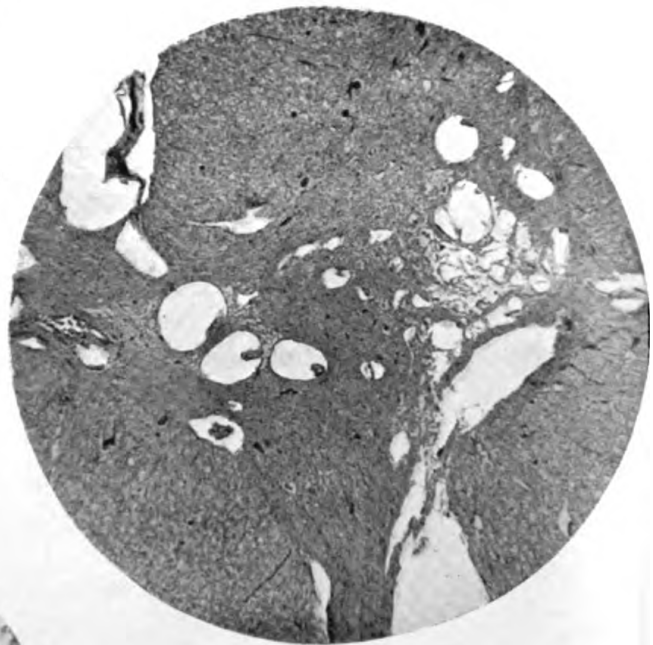


FIG. 2. Showing an atrophic condition in one portion, also numerous rounded cavities with vessels attached to one side.

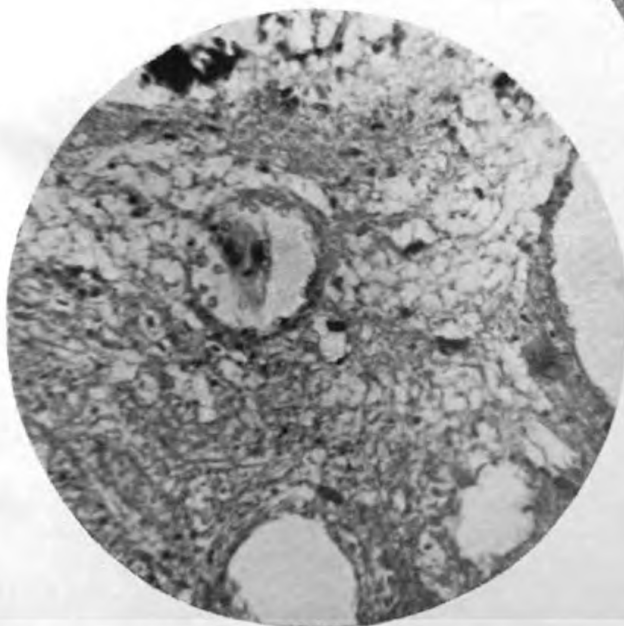


FIG. 3. Showing walls of cavities formed of neuroglia fibres pressed together, and an advanced degree of atrophy of nerve elements and neuroglia.



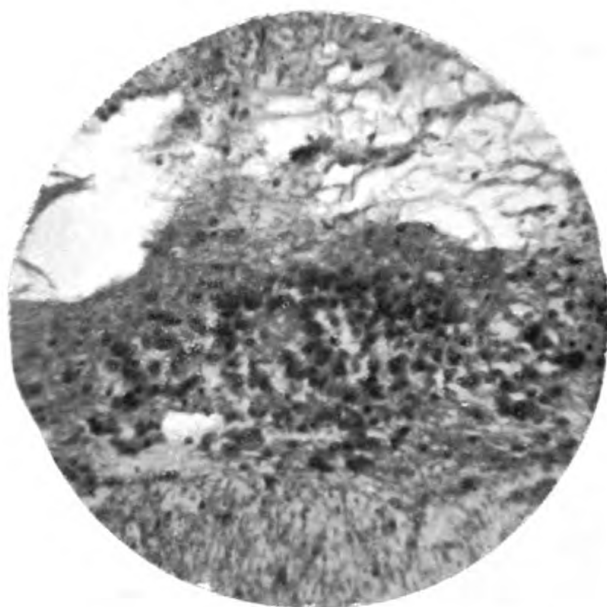


FIG. 4. Showing central canal filled with nucleated cells, with atrophic condition in front, and the tips of the posterior columns posteriorly.



FIG. 5. Showing vessel with thickened, hyaline-degenerated wall. Lumen almost occluded at one point.



FIG. 6. Showing on the right an artery with deeply stained muscular coat and hyaline degeneration of perivascular sheath; and on the left a vein with hyaline-degenerated wall.

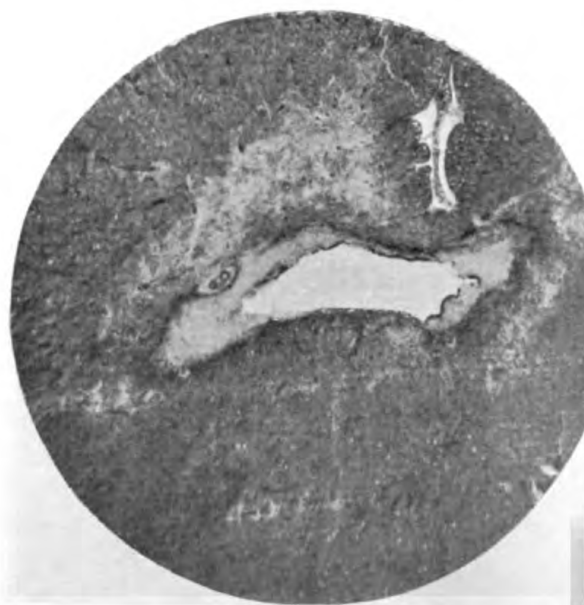


FIG. 7. Showing dilated central canal lined with columnar epithelium, with rarefaction around and near it.





FIG. 8. Showing large cavity occupying the grey matter on both sides of the cord.

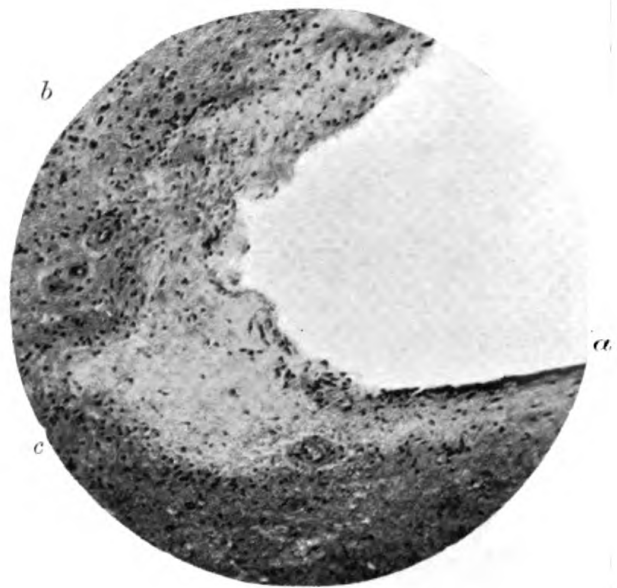


FIG. 9. Showing portion of the wall of dilated central canal: (a) columnar epithelium; (b) neuroglial nuclei; (c) process of rarefaction progressing.

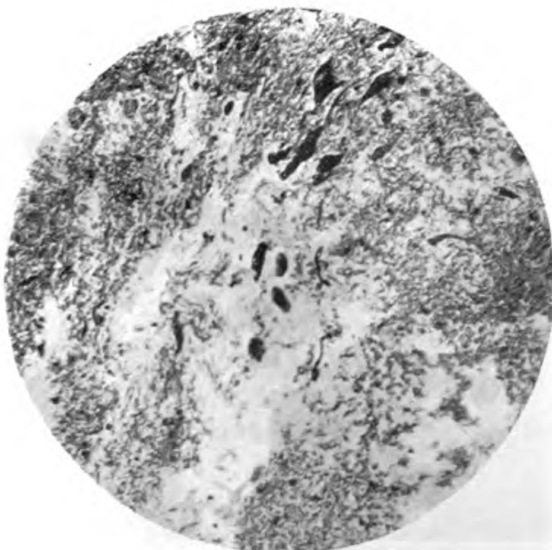


FIG. 10. Showing tissues broken down by hæmorrhage, and forming a cavity in the grey matter.

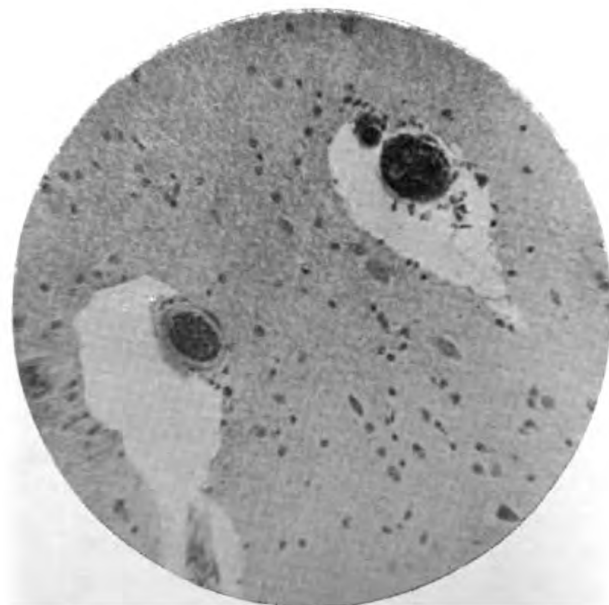


FIG. 11. Showing spaces around vessels. Notice the absence of overgrowth of neuroglia.





## NOTE ON THE TENDO-ACHILLES JERK AND OTHER REFLEXES IN DIABETES MELLITUS.

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MUCH attention has been recently devoted to the tendo-Achilles jerk in nervous diseases, and many observations have now shown that this reflex may be lost before the knee-jerk at a very early stage of locomotor ataxia.

I have recently examined the tendo-Achilles jerks in fifty cases of diabetes mellitus, and have found both absent in nineteen. It is well known that the knee-jerks are often lost in severe forms of diabetes, and when the knee-jerks have been absent in diabetes I have usually found the tendo-Achilles jerks lost also.

But it is interesting to note, that in diabetes mellitus, as in locomotor ataxia, the tendo-Achilles jerks may disappear before the knee-jerks are lost. Thus in eight cases of diabetes in which the tendo-Achilles jerks reflexes were lost, the knee-jerks were present. In tabular form, the results of the examination of the tendo-Achilles jerks and knee-jerks in fifty cases of diabetes were as follows:—

	Cases.
(A) Tendo-Achilles jerks both absent in . . . . .	19
In these cases <i>knee-jerks both present</i> in . . . . .	8
One knee-jerk absent, one present in . . . . .	3
Both knee-jerks absent in . . . . .	8
(B) One tendo-Achilles jerk absent, one present in . . . . .	2
In these cases both knee-jerks present.	
(C) Tendo-Achilles jerks both present in . . . . .	29
In these cases both knee-jerks present in . . . . .	28
One knee-jerk present, one absent in . . . . .	1

The knee-jerks are often lost in severe forms of diabetes, especially in hospital patients; whilst amongst private patients, with better conditions of life, the knee-jerks are lost less frequently.

In 100 cases of diabetes, nearly all of whom were *hospital* patients, I found the condition of the *knee-jerks* as follows:—

	Cases.
Both jerks lost in . . . . .	49
One present, one absent in . . . . .	6
Both present in . . . . .	45
	<hr/>
	100

Amongst 50 cases of diabetes recently examined in *private practice* I found:—

	Cases.
Both knee-jerks lost in . . . . .	6
One present, one absent in . . . . .	1
Both present . . . . .	43
	<hr/>
	50

I have met with five cases of perforating ulcer of the foot in diabetes mellitus, resembling the form which occurs in *tabes*; in three of these cases the knee-jerks were absent, in one case both knee-jerks were present, and in one case one knee-jerk was present and the other absent.

In severe cases of diabetes the *wrist-jerks* are often absent; usually they are lost when the knee-jerks are absent.

In 50 cases (mostly of the severe form) the condition of the wrist-jerks was as follows:—

	Cases.
Both absent in . . . . .	30
„ present in . . . . .	19
One absent, one present in . . . . .	1

It is to be remembered that the wrist-jerks are sometimes absent in healthy individuals. In the examination of over 100 individuals, who were either in good health or were suffering from some local surgical affection not likely to have any influence on the reflexes, I found the wrist-jerks present in 75 per cent., absent in 25 per cent.

In the 19 cases of diabetes in which the wrist-jerks were present, the knee-jerks were also present in 18. In the 30 cases in which the wrist-jerks were lost, the knee-jerks were absent in 25.

The superficial reflexes—plantar, abdominal and epigastric—are probably as frequently present in diabetes as in health. In the severe forms of diabetes, when the knee-jerks are absent the superficial reflexes are generally present, and the *abdominal* and *epigastric reflexes* are usually much *increased*.

The plantar reflex is of the normal flexor type.

In cases in which the knee-jerks are lost, I have not found any evidence of muscular hypotonus, which is so common in tabes.

## Abstracts

### ANATOMY.

**THE ANTHROPOLOGY OF THE SPINAL CORD.** H. PFISTER, (327) *Neurolog. Centralbl.*, 1903, p. 757, 819.

THE author has made a series of observations in children on the relative weight of the spinal cord to the brain and body weight, and the alteration of these relations during development. The following are the results obtained :—

1. The spinal cord of the boy is at all ages, on the average, heavier and longer than that of the girl.
2. In comparison to the brain, the spinal cord of the boy at birth is lighter than that of the girl. In children of the same age and same sex a heavy brain is accompanied by a heavy spinal cord. During life the proportion  $\frac{\text{weight of spinal cord}}{\text{weight of brain}}$  alter in a similar manner in both sexes. The proportion in a new-born child is  $\frac{1}{10}$ , whilst in the adult it is  $\frac{1}{8}$ . From the seventh to tenth month of life the proportion is  $\frac{1}{7}$ , and in the seventh year  $\frac{1}{6}$ .
3. The spinal cord weighs at birth from 3 to 3·4 grammes, and increases about eight times its weight (27 to 28 grammes), this increase being most marked during the first two years of life, becoming less as years advance.
4. In new-born children about 1 gramme of weight of spinal cord corresponds to about 14 cm. of body length. As age advances the proportion alters, so that in the adult 1 gramme in weight of spinal cord corresponds to about 6·2 cm. of body length.

5. The average length of the spinal cord in the new-born child is about 14 cm., and increases about three times its length in adult life (45 cm.).
6. In the new-born child the relative length of the spinal cord to the length of the body is about 29.5 per cent., but at the end of the first year this falls to 26 to 25 per cent.

The above figures form a useful basis for further observation on the weight of the spinal cord in children.

The cords were weighed without dura mater, and without the roots of the cauda equina.

FREDERICK E. BATTEN.

**ON THE SPECIAL FIBRILLAR APPARATUS IN THE NERVE  
(328) CELL ELEMENTS OF CERTAIN CENTRES OF THE  
ACOUSTIC.** ARTURO DONAGGIO, *Rivista Sperimentale di  
Freniatria*, 1903, f. i.-ii.

THE author has applied his methylene blue methods to the study of the histological features of the nervous elements of the acoustic centres, and has observed in certain cells of the ventral nucleus a special character of the endocellular fibrillar apparatus, of which he gives a minute description in this paper. The cells in question evidently correspond to the unipolar cells described by L. Sala in this nucleus. They are of medium size, compared with the other cells of the nucleus, and are round or oval in form, but somewhat irregular in contour. In these cells the endocellular fibrillar apparatus, which was first described by the author in 1896, does not pervade the whole cell, but forms a nest in the deeper part, leaving a broad clear zone at the periphery. The stain is selective for the neurofibrils; the nucleus, which the fibrillar apparatus surrounds, is not stained. These cells have one prolongation, the axis-cylinder process. The fibrils extend into this process, which becomes a nerve fibre of the corpus trapezoideum. The unipolarity is, strictly speaking, relative to the form of the endocellular reticulum, for some of the cells, at least, have short processes. The author has observed a similar character of the endocellular fibrillar apparatus in some of the cells of the nucleus of the corpus trapezoideum. He thinks it is probable that these particular cells are the same as the unipolar cells described by Veratti in this situation. Veratti maintains that the pericellular baskets—the acoustic terminations of Held—which are generally regarded as the result of the subdivision of the large nerve fibres around the cells, really represent the membrane of the unipolar cells incompletely stained, and that the large fibres are not directed to the nerve cells and do not form arborisations around them, but are the axis-

cylinders of these cells. Donaggio has, however, been able, with the aid of one of his methods, to determine that the axis-cylinders of the unipolar cells and the large nerve fibres are distinct. The latter are each composed of a very large number of longitudinal fibres which, at a varying distance from the cell, break up into numerous bundles like tufts of hair, which come into relationship with the cell. The exact relation of these ramifications to the cell-body he describes in another paper (see page 683).

W. FORD ROBERTSON.

**THE FIBRILS IN THE NERVE CELLS OF MAMMALS.** ARTURO  
(329) DONAGGIO, *Gazzetta medica italiana*, N. 44-45, 1903.

IN this paper the author briefly states the present position of the question as to the nature of the fibrillar structure contained in the nerve cells, to which he has himself made an important contribution with the aid of his methylene blue method. He maintains that the view of Bethe, that in vertebrates the nerve fibrils traverse the cell-body without forming a network, is based upon the evidence of preparations in which the fibrils are incompletely stained. In preparations by his own method very numerous delicate fibrils are to be observed, forming by their anastomoses a dense network. He mentions the following new points that he has recently determined regarding this fibrillar structure. The arrest of the network around the nucleus, and the absence of any trace of colour in the nucleus, in well differentiated preparations, are constant features. The fibrillar network has generally a closer character towards the centre of the cell. The undivided fibrils occur almost exclusively at the periphery, and they are much more numerous and delicate than Bethe described them to be. Donaggio considers that many of the recent hypotheses regarding the function of the nerve cell rest upon an incomplete basis of fact, and, in particular, that the theory of Bethe regarding the functional value of the nerve cell and the course of the stimuli is clearly ill-founded.

W. FORD ROBERTSON.

**FURTHER OBSERVATIONS ON THE NATURAL MODE OF  
(330) SUBDIVISION OF THE MAMMALIAN CEREBELLUM.**

G. ELLIOT SMITH, *Anatomischer Anzeiger*, Bd. xxiii. Nos. 14 and 15, July 30, 1903, pp. 368-384, with 25 figures.

THE object of this communication is to elaborate and perfect the tentative scheme for subdividing the cerebellum which the author put forward in 1899 ("The Brain in the Edentata"). The present scheme is based upon the results obtained from a study

of the cerebellum in practically every mammalian genus and from an investigation of its developmental history in representatives of the Marsupialia, Insectivora, Rodentia, Ungulata, Carnivora, and Primates. The need for an exceptionally wide basis for any scheme of cerebellar subdivision (which is to be a natural expression of the morphology of the organ) is of paramount importance. The reason for this is that homologous fissures make their appearance in the developmental history of the cerebellum in very different chronological orders in different genera and species, or even in different specimens of the same species, so that all such data must be checked by a reference to the great body of facts culled from as wide a source as possible. The neglect of this precaution has led to some very curious results in the recent memoirs of several writers.

The most primitive type of mammalian cerebellum, that of the Marsupial Mole, is described in detail. It consists of three simple azygos leaves—anterior, middle, and posterior lobes—and two simple floccular lobes. The process of progressive complication of these lobes is then described in the great body of mammals. The posterior lobe (which even in the Marsupial Mole is already subdivided into nodulus and uvula) never attains to large proportions. The anterior lobe becomes subdivided at a very early stage; but the most noteworthy expansion and complication occurs in the lateral parts of the middle lobe, which alone is subdivided into vermis and lateral “lobes” or *alæ*. The only constant subdivision of the vermis (of the middle lobe) consists of the separation of a *pars pyramidalis* from a *pars suprapyramidalis*. This subdivision extends to the *alæ*. It is only in the highest Primates that the *parapyramis*, *i.e.* that part of the *ala lobi medii* which is alongside (and joined to) the pyramid, becomes split up into biventral and tonsillar parts, so that the new term “*parapyramis*” becomes necessary in the case of most mammals. Moreover, in the vast majority of mammals the homologue of the tonsil is joined, not to the uvula, but to the pyramid.

That part of the *ala lobi medii* which is joined to the *pars suprapyramidalis vermis*, becomes split up (in most mammals) into three parts, an anterior “*area lunata*” (representing Koelliker’s “*lobulus lunatus posterior*”); a middle “*area pterioidea*” (so called because in most mammals it exhibits a very distinctive feather-like pattern of *folia*); and a posterior “*area postpteroidea*.”

In many mammals (including most small mammals, all the Edentata, Carnivora, and the Lemurs) the *area postpteroidea*, together with the *parapyramis*, forms a very characteristic vertical worm-like band alongside the vermis. As it is, for descriptive purposes, desirable to have a name for this, I have called it the “*paravermis*.”

AUTHOR'S ABSTRACT.

**THE CENTRAL NERVOUS SYSTEM OF THE CETACEÆ. I. The**

(331) Spinal Cord of *Phocoena communis*, Cuv, and the Cervical Region of the Cord of *Balaenoptera rostrata*. BERNARD RAWITZ, *Arch. f. mikr. Anat.*, Bd. 62, H. 1, S. 1.

UNTIL recently no complete description has been given of the minute anatomy of the spinal cord of any of the Cetaceæ, largely on account of the difficulty in procuring material in a suitable condition, but the musculature of this class of mammals presents such striking modifications that a careful comparison between the cord of any member of this group and that of man or any quadruped would be of great interest.

This account is fairly complete, but the writer institutes few comparisons, and the result is rather disappointing. The chief points of general interest are as follows. The central canal was obliterated, and there was marked asymmetry throughout between the right and left sides of the cord.

The author does not give an account of the appearance found at each segment, as should be done in every description which claims to be complete, but he finds evidence of marked alteration in the shape of the grey matter at different levels.

In the lower cervical region the anterior horns become enlarged laterally, while the anterior part is diminished.

The dorsal region can be divided into two parts, an upper and a lower, and the same division exists in the lumbar region.

In the lumbar region an unpaired mesial dorsal horn of grey matter appears, probably representing Clarke's column, and, lower down, isolated masses of grey matter are found in the dorsal columns.

The illustrations are fairly good, though rather diagrammatic, and the author makes no reference to the important work by Hotschek on the spinal cord of the dolphin published in 1898.

DAVID WATERSTON.

**ON THE COMPARATIVE ANATOMY OF THE CORPUS STRIATUM**

(332) (AVIAN BRAIN). L. EDINGER, "Comptes rendus de l'association des Anatomistes," V<sup>e</sup> Session, Liege, 1903.

THIS valuable paper shortly summarises, and in part expands, the work on the bird's forebrain abstracted in an earlier number (Abstract 135).

Though our knowledge of the avian brain is scarcely strong support to the hypothesis of a continuous cerebral development in the animal series, the relatively enormous bulk of its corpus striatum with the insignificant development of the cortex and its fibre-systems, makes it a suitable subject for such investigation of the structure and anatomical relations of the basal forebrain

ganglia as may later form a basis for the determination of their physiological functions.

Here, as in the other vertebrate classes, there is no direct connection between the pallium and corpus striatum visible, but the massive tract which binds the latter to the thalamus, as described by Edinger in the lower vertebrates as well as in mammals, is again found. As its fibres run in each direction it is a double connection.

The chief mass of the corpus striatum consists of the dorsally lying hyperstriatum which stretches the whole length of the fore-brain. It contributes liberally to the tractus strio-thalamicus and is also connected to the midbrain nuclei by large tracts. Its importance is indicated by the fact that it contains association fibres of its own.

Ventral to this ganglion lies the crescentic-shaped mesostriatum, whose large frontal end appears laterally on the ventral surface of the brain as the nucleus ventralis anterior (nucleus basalis), and mesially forms the lobus parolfactorius. The former nucleus is of special interest as it sends fibres to the medulla oblongata, and from Kalischer's physiological experiments seems to be part of the central apparatus for deglutition.

A third ganglion, the epistriatum, which lies laterally in the occipital portion of the brain, receives a large tract from the lobus parolfactorius and is connected with its fellow by the anterior commissure, which, as the olfactory apparatus is rudimentary, must be chiefly composed of interstitial fibres.

A homologous ganglion with similar connections has already been demonstrated in the fish and reptilian brains.

A fourth ganglion is the ectostriatum, a wedge-shaped structure whose edge is inserted laterally between the hyperstriatum and mesostriatum, which sends an important tract to the midbrain.

This ganglion cannot be homologised with any known structure in the reptilian or mammalian brain and seems characteristic of the avian.

GORDON HOLMES.

## PHYSIOLOGY.

### MUSCULAR MOVEMENTS AND THEIR REPRESENTATION IN (333) THE CENTRAL NERVOUS SYSTEM. *The Croonian Lectures.*

CHARLES E. BEEVOR, M.D. Lectures III. and IV.

#### LECTURE III.

*Movements of the head.*—The sterno-mastoids are the chief flexors of the head and act in conjunction with the platysma myoides, the omohyoids, and the other depressors of the hyoid bone. Extension of the head is carried out by the clavicular fibres of the trapezii, the complexi splenii capitis, and probably the trachelo-mastoids. The question as to whether the sterno-



mastoids ever act as extensors in any position of the head is answered by Dr Beevor in the negative.

*Extraordinary muscles of respiration.*—In dyspnoea the extensors of the head fix the skull, and then the sterno-mastoids act as inspiratory muscles. The pectoralis major does not help in inspiration, and the action usually attributed to that muscle is really performed by the pectoralis minor. The serratus magnus, the scaleni and the claviculo-occipital fibres of the trapezius are accessory muscles of inspiration.

The latissimus dorsi is an extraordinary muscle of both inspiration and expiration; in the former act its lower fibres serve to elevate the lower ribs; in the latter it both compresses the posterior part of the abdomen, and so fixes the lower ribs that the external oblique muscles, with which it interdigitates, are brought into action to their best advantage.

The movements of the lower limbs and of the facial and ocular muscles are not dealt with.

The views of Galen, Winslow, Hunter and Duchenne, and the more recent experimental work of Demeny and Sherrington, all bearing on the part played by the antagonistic muscles in any particular movement, are fully discussed in this lecture, and the conclusions arrived at may be thus summarised:—In all movements against extreme resistance or against gravity the muscles antagonistic to these movements do not act; on the other hand, in unopposed movements where gravity does not come into play, as in rotation of the head, the antagonistic muscles are again found not to contract. In so-called functional or hysterical paralysis it is not unusual to find that during the attempt to perform a simple voluntary movement the first muscle to contract is the one which is directly antagonistic to the movement desired.

Loss of a particular movement, when the muscle which usually performs it is able to carry out other movements, is sometimes seen in cases of cerebral palsy. In some hemiplegics, for instance, the biceps can flex the elbow but cannot supinate the forearm; in others the movement of elevating the shoulder is lost, while that of adducting the head towards the paralysed arm is capable of being carried out, although the same muscles are involved in both actions. Dr Jackson first called attention to the paralysis of the clavicular fibres of the trapezius in elevating the shoulder in hemiplegia, although the muscle retained its power of acting bilaterally in deep inspiration. A similar phenomenon is found in connection with the latissimus dorsi, which has three actions, viz.: (a) acting bilaterally in sneezing and reflex coughing; (b) acting bilaterally in voluntary coughing; and (c) acting unilaterally in movements of the arm. In hemiplegia of cerebral origin it is usual to find that the muscle comes into action in (a) and (b), but not in (c) on the paralysed side.

Another point of interest is raised by the apparent selective paralysis of the clavicular fibres of the pectoralis major for one movement and not for another, in lesions of the brachial plexus or cervical cord. These fibres in the healthy person are brought into action with the other parts of the muscle in adducting the arm towards the middle line; they are also associated with the deltoid muscle in advancing the humerus.

In the cases referred to it was found that the clavicular fibres contracted in the attempt to adduct the arm, but not in the attempt to advance the humerus, the deltoid being paralysed as well. Dr Beevor is unable to offer a satisfactory explanation of this curious fact.

*The sequence in which various muscles enter into a movement with varying degrees of resistance to be overcome.*—All the muscles which are grouped together for the performance of a movement do not come into action when only slight effects are required, and a certain increase of work has to be encountered before they all act. On the other hand, besides the muscles directly concerned in a movement, other muscles are put into action if the resistance is excessive; for instance, the flexors and extensors of the elbow are associated with the forearm muscles in such movements as flexing the fingers, flexing the fingers and thumb, flexing and extending the wrist when a considerable degree of power is desired. The amount of resistance required to call in these auxiliary muscles was measured by Dr Beevor, both when the forearm was supinated and when it was pronated, with the results represented in this table.

*Table showing the Sequence of Movements in the Upper Arm to Movements of the Hand in Positions of Supination and of Pronation.*

Movement performed.	Position of the forearm.	Amount of work required to be done before the triceps contracts.	Amount of work required to be done before the biceps contracts.
Fingers flexed—Traction at right angles to the line of the forearm . . .	Supinated.	No contraction.	22 pounds.
Fingers extended . . .	Pronated.	1½ pounds.	No contraction to 18 pounds.
Thumb extended . . .	Supinated.	4 pounds.	No contraction.
Thumb and fingers flexed (grasping) . . .	Pronated.	No contraction.	„ „
Thumb and fingers flexed (grasping) . . .	„	„ „	„
Wrist flexed . . .	Supinated.	24 pounds.	33 pounds.
„ „ . . .	Pronated.	13 pounds.	30 pounds.
Wrist extended . . .	Supinated.	No contraction to 32 pounds.	22 pounds.
„ „ . . .	Pronated.	2 pounds.	No contraction.
„ „ . . .	Supinated.	4 pounds.	No contraction to 24 pounds.
„ „ . . .	Pronated.	No contraction.	22 pounds.

One of the questions which arises from this investigation is:— Why in the supinated position of the forearm does the triceps not co-operate until the grasp has reached 24 lbs., whereas in the pronated position it takes part in the movement when the grasp has reached 13 lbs.? It must mean that in the position of pronation the forearm muscles produce flexion of the elbow sooner than in the position of supination, and therefore that the triceps must intervene sooner to prevent this flexion. If this be true it is in harmony with the fact that patients who have lost their proper flexors of the elbow manage to produce the movement, after first putting the forearm into the position of pronation.

#### LECTURE IV.

A classification of the various muscles which take part in a single movement under different titles has been attempted before by Winslow, Duchenne and others, but Dr Beevor has drawn up a new scheme under which he describes: (i.) prime movers; (ii.) synergic muscles; (iii.) fixation muscles, (a) indispensable and (b) postural; (iv.) antagonist muscles in occasional movements.

For example, in grasping an object, the *prime movers* are the flexors of the fingers and thumb, and the small thenar muscles; the *synergic muscles* are the extensors of the wrist; and the *indispensable fixation muscles* are the triceps and biceps.

The part taken by the various muscles in a movement is unknown to the will and, as John Hunter pointed out, we cannot throw into action a single muscle separately and independently of the collateral effects of others. In the words of Dr Hughlings Jackson, "nervous centres know nothing of muscles, they only know of movements."

A good example of the inability of the will to supplement loss of power in certain muscles by the employment of others is afforded by the cases of lead palsy already referred to where the extensors of the wrist will not extend that joint except when the hand is closed.

It appears impossible, too, except by long training, for the will to leave out a muscle from a combination to which it naturally belongs, or to add to a combination a muscle with which it is not naturally associated. On the other hand, it appears possible, as recommended by Nicalodoni, to take a muscle from one group and to introduce it into another by the transplantation of tendons.

The remainder of this lecture is devoted to discussing the probable site of the co-ordination which evidently takes place somewhere in the nervous system when any movement involving the action of a combination of muscles is performed voluntarily.

There is considerable evidence to show that the anterior horn

cells in the spinal cord are arranged on a morphological rather than a physiological basis, and that the fibres contained in the cords of the brachial and of the lumbo-sacral plexus are distributed according to an anatomical scheme.

On the other hand, it has been proved that co-ordinated reflex movements can be produced by stimulation of posterior root fibres, even when the spinal cord is cut off from connection with the higher centres. Again all experimental and clinical evidence goes to show that the cerebral motor cortex is the seat of the representation of movements and not of individual muscles.

In the present state of our knowledge it is impossible to say definitely whether the origin of this muscular co-ordination is situated in the cerebral cortex or whether, as Monakow has suggested, there are intermediate cells situated between the spinal endings of the pyramidal fibres and the cells of anterior horns, which are connected in function with the associated action of muscles.

Some researches which were undertaken by Sir Victor Horsley and Dr Beevor in monkeys with the object of ascertaining whether the movements obtained on the homolateral side of the body, when the cortex was stimulated, were produced through the corpus callosum and opposite hemisphere, or through the commissural fibres in the spinal cord, seem to favour the former alternative. This result is in opposition to the conclusions arrived at by Francois-Franck experimenting on cats and dogs.

E. FARQUHAR BUZZARD.

**NEW INVESTIGATIONS ON SPINAL LOCALISATION.** PARRON (334) and C. PARRON, *Journ. de Neurol.*, June 20 and July 5, 1903, p. 263.

THE results furnished in this paper were obtained by extirpation of the individual muscles in dogs and subsequent examination of the spinal cord. A definite group or groups of cells was in each case found in reactionary tigrolysis, and the conclusions applied to the human cord were arrived at by seeking out the homologous groups at or about the corresponding level.

*Sterno-mastoid.*—Innervated from a central group of cells from the junction of the bulb and spinal cord to 2 C. There is a similarly situated group in the first two cervical segments in man.

*Biceps brachii.*—A postero-internal group in 6 C. In man in 5 C.  
*Brachialis anticus.*—A postero-lateral group in 6 C. In man in 5 C.

*Flexor sublimis digitorum.*—An antero-central group in 7 C and 8 C.

*Extensor carpi radialis*.—From the postero-external angle of the ventral horn in 8 C.

*Spinalis dorsi*.—Though all previous investigations lead to the conclusion that the spinal muscles are innervated from columns of cells placed antero-internally in the ventral horns, the only group of cells found in reaction after extirpation of this muscle lay in the ventral part of the horn in the first four dorsal segments, near, but not in, the antero-internal angle.

*Longissimus dorsi*.—A similarly placed group from 5 D to 4 L.

*Sartorius*.—An antero-external group in 3 L and 4 L. In man in 3 L.

*Quadriceps femoris*.—From the external angle of ventral horn in 3 L and 4 L.

*Adductor longus*.—An antero-central group in 3 L and 4 L.

*Adductor magnus*. } Together form a postero-central group in  
*Adductor brevis*. } 3 L and 4 L.

*Gracilis*.—A meso-central group in 3 L.

The localisation of the spinal motor supply of several other muscles as already demonstrated by various other workers are referred to, and these results taken with their own are regarded by the authors as showing that the localisation in the ventral horns is muscular, that each muscle has a centre for itself in the spinal cord. But while large muscles with single functions have centres apart from others, groups of muscles with common functions have centres grouped more or less together, so that the arrangement of the motor cells in the ventral horns is also functional, that is, each independent function has an isolated spinal centre.

GORDON HOLMES.

**THE FUNCTIONS OF THE FRONTAL LOBES** (2 figs. and 1 plate).  
 (335) JOSEPH SHAW BOLTON, *Brain*, Part cii., Summer 1903.

THIS paper deals with the subject of the functions of the frontal lobes from the standpoints of the morbid anatomy of mental disease, and the general histology of the cerebral cortex in normal individuals, and in the subjects of mental disease.

The present position of the question from the experimental and clinico-pathological points of view is first discussed, and, when dealing with the "psycho-motor" area, it is suggested that this region of the cerebrum may be strictly neither "motor" nor "sensori-motor" in function, but may be homologous with the lower associational areas of the cortex. This area is now known to occupy a much less extensive region of cortex than was at first defined, and it is not improbable that it bears to some projection

area, as yet undefined, a similar relationship to that existing between, *e.g.* the visuo-psychic and the visuo-sensory regions.

After due reference to the literature dealing with the functions of the prefrontal lobes, the researches of Flechsig are referred to and discussed at some length.

The subject-matter of the paper is now reached, and in it the writer brings forward evidence to prove that *the anterior centre of association of Flechsig is the region concerned with attention and the general orderly co-ordination of psychic processes*, and that *the cellular elements throughout the cortex which are especially concerned in the performance of associational functions are those of the pyramidal layer of nerve cells*.

The evidence which is adduced is derived from the researches which have been conducted by the writer during the past seven years, and it is considered under the heads of (1) *Morbid Anatomy* and (2) *General Histology*.

1. MORBID ANATOMY OF MENTAL DISEASE.—The especial object of the writer has been to correlate the morbid anatomy and the clinical features of the different types of mental alienation, this being a subject on which relatively little care has hitherto been bestowed. He has demonstrated, in 200 cases of mental disease, that the amount of cerebral wasting, and the associated morbid changes inside the cranium in these cases, vary directly with the amount of dementia (*i.e.* permanent psychic disability) existing in the patients. This relationship, with certain restrictions which are referred to, is much more absolute than might appear probable, owing, in the majority of cases of insanity, to a more or less complete removal of the products of degeneration by the time of death. In this connection certain differences which exist between the morbid anatomy of ordinary dementia and that of dementia paralytica are referred to at length.

*The regions of wasting in dementia*, considered generally (for individual variations exist) are, from observation of several hundred cases, as follows:—(1) The greatest amount occurs in the pre-frontal region (anterior two-thirds or so of the first and second frontal convolutions, including the neighbouring mesial surface, and the anterior third or so of the third frontal convolution).

(2) The wasting is next most marked in the remainder of the first and second frontal convolutions. [In dementia paralytica Broca's convolution should, as a rule, be included here and (2) and (3) should follow (4).]

(3) It is, perhaps, next most marked in the ascending frontal and Broca's convolutions, though this grade should, in many cases at least, follow (4).

(4) It is next most marked in the first temporal convolution and the insula, and in the superior and inferior parietal lobules.

In practically all cases it is more marked in the two former than in the two latter.

(5) It is least marked in the remainder of the cerebrum (including the orbital surface of the frontal lobes), particularly the inferio-internal aspect of the temporo-sphenoidal lobe and the posterior pole of the hemisphere.

Apart from the necessarily excluded abnormalities of development, which are of vascular or traumatic origin, *the degrees of under-development in amentia* follow the order given above, at least as regards (1) and (2).

It is concluded from the above that the great anterior centre of association is the region of the cerebrum which is primarily affected in mental disease, all the neighbouring or bordering regions being concerned to a less extent, probably from chronic atrophy of the related systems of fibres of association.

2. GENERAL HISTOLOGY (NORMAL AND MORBID) OF THE CEREBRAL CORTEX.—The writer repeats the thesis, which he has already advanced in previous papers, that the cortex cerebri, in the regions referred to as the centres of association of Flechsig, consists in the adult of five primary layers. Of these, two, the first or superficial and the fourth, are essentially nerve fibril layers; the remaining three, namely the second, third and fifth, containing respectively the pyramidal, the granule and the polymorphic cells, are essentially nerve-cell layers.

The following conclusions are drawn from his researches:—

(1) *The second or pyramidal layer of the cortex cerebri.*—(a) *The prefrontal region.*—The pyramidal layer is the last cell-layer of the cortex to develop during the process of lamination, and it is also the first to undergo retrogression in dementia. It is the only layer which appreciably varies in depth in normal brains; the degree of its development in normal foetuses and infants, and in aments of every grade from the idiot to the chronic or recurrent lunatic without dementia, varies directly with the mental power of the individual; and the degree of its retrogression in demented patients varies directly with the amount of dementia existing in the respective patients. (b) *The visuo-psychic region.*—The pyramidal layer reaches practically the same adult depth as in the prefrontal region, but it does not vary in depth according to the degree of dementia, though a small and practically constant decrease in depth is evident. This layer develops much later than does the pyramidal layer in the visuo-sensory region, and in a child of one month it is less than two-thirds of the adult depth. (c) *The visuo-sensory region.*—The pyramidal layer in this region is in the adult only about five-ninths of its depth in the regions above referred to. It, however, develops much earlier, being in infants of one and three months very little below the adult depth.

*The pyramidal layer, therefore, subserves the "psychic" or associational functions of the cerebrum. This is pre-eminently the case in the prefrontal region, less so in the visuo-psychic region, and least of all in the visuo-sensory region. These three regions are therefore of different grades in the hierarchy of cerebral function.*

(2) *The third, or granule layer, is developed before the pyramidal layer. In the primary visual area the optic radiations end in the midst of the hypertrophied and duplicated granule layer. This layer, therefore, probably, reasoning by analogy, subserves the reception or immediate transformation of afferent impressions, whether from the sense-organs, or from other parts of the cerebrum.*

(3) *The fifth or polymorphic layer is the first layer to be differentiated during the process of lamination, and it is the last to fail in the retrogression of dementia. A decrease in this layer exists in extreme aments (normal or otherwise), and in demented who are unable to carry on the ordinary animal functions, such as attending to their own wants, etc. This layer, therefore, probably subserves these lower voluntary functions of the animal economy.*

*The writer hence considers it proved that the great anterior centre of association lying in the prefrontal region is underdeveloped on the one hand in all grades of primary mental deficiency, and on the other undergoes primary atrophy pari passu with the development of dementia; it is therefore the region of the cerebrum which is concerned with the performance of the highest co-ordinating and associational processes of mind.*

AUTHOR'S ABSTRACT.

#### ON THE CORTICAL CENTRE FOR DEVIATION OF THE HEAD.

(336) SCHUPFER, *La Rif. Med.*, No. 27, 1903.

THE following case is of interest because of a small lesion which was limited to the posterior part of the second frontal convolution. This lesion was caused by a sword wound which produced a depression in the left parietal bone. For about twenty days after the injury there was deviation of the head to the right. The patient could only move his head a very little back towards the middle line; passive movement of the head was also very limited. This deviation gradually disappeared. The movements of the eyes during this period were normal. At the post-mortem examination a prominence was found on the inner side of the left parietal bone, the pia mater was thickened and opaque below this, and the root of the left second frontal convolution was diminished in size and in consistency, and had a gelatinous aspect. Around this area of softening there was some cicatricial tissue which extended to the white substance below. Various facts suggested that there had been an irritative lesion at first, and that



the softening took place subsequently. After giving, in some detail, the results which have been obtained by numerous experimenters by irritation and destruction of this region, the author offers the following conclusions:—

1. That following an irritative circumscribed lesion of the frontal lobes which has its area of maximum intensity at the foot of the second frontal convolution, deviation of the head towards the side opposite the lesion may be the only sign present.

2. This deviation is temporary.

3. That a destructive lesion of the foot of the second frontal convolution does not, after a short time, cause any deviation of the head and eyes.

4. That probably the centre or one of the centres, for deviation of the head in man, has a position analogous to that observed in monkeys.

5. That probably the centre for deviation of the head is independent of that for deviation of the eyes.

The author also draws attention to the fact that with this destructive lesion of the foot of the second frontal convolution there was no agraphia or disturbance of the power of writing.

R. G. Rows.

**A HISTO-PHYSIOLOGICAL QUESTION REGARDING THE  
(337) NERVOUS TRANSMISSION BY CONTACT BETWEEN  
THE ACOUSTIC TERMINATION OF HELD AND THE  
CELLS OF THE NUCLEUS OF THE CORPUS TRAPE-  
ZOIDEUM. ARTURO DONAGGIO, *Rivista Sperimentale di Freni-  
atria*, 1903, f. i.-ii.**

THE author first refers to the various opinions that have been expressed regarding the nature of the acoustic terminations of Held, described in the nucleus of the corpus trapezoideum in 1892. Ramon y Cajal, on the ground that the nests or chalices of Held envelop the cells without penetrating them, has regarded these terminations as constituting one of the most demonstrative examples of a connection by contact between the nervous elements, and one of the most decisive proofs that can be adduced against the opinion of those who doubt the possibility of the passage of the nervous current from a fibre to a nerve cell across the interstitial substance. Held, in a later paper, defended his contention that the ramifications are, in the adult at least, adherent to, and fused with, the periphery of the cell. Veratti has demonstrated the presence of unipolar as well as multipolar cells in the nucleus in question, and interprets the chalices as the products of the partial impregnation of a membrane which surrounds the

unipolar cells; he holds that the large nerve fibres are the axis-cylinder processes of these cells.

Donaggio has applied to the investigation of this question his methylene blue method for the demonstration of the peripheral reticulum of the nerve cell, and also that for the staining of his endocellular reticulum. The material used was obtained from dogs, rabbits, cats and guinea-pigs. The chief interest attaches to the facts ascertained by means of the application of the method for the coloration of the endocellular reticulum. A clear demonstration was obtained of the fibrils of the large nerve fibres as well as of those composing the endocellular reticulum of the unipolar cells. The relationship of the two was ascertained to be as follows:—The large nerve fibres ramify and the fibrils composing them assume the form of small bundles, which in turn separate more or less quickly according to their distance from the nerve cells. The fibrils neither remain external to the nerve cell nor fuse with its periphery. Some of them are inserted into projecting portions of the clear peripheral zone, but remain distinguishable from it. After coursing in this peripheral zone for some distance, they dip down and reach the endocellular fibrillar apparatus with which they are directly continuous. Others pass straight across the peripheral clear zone to the endocellular fibrils. From these observations it would appear that the acoustic terminations of Held are not really nerve fibril terminations, but a part of a rich fibrillar conducting system continuous in nerve fibre, ramification and nerve cell.

W. FORD ROBERTSON.

### **PATHOLOGY.**

**A CASE OF ANENCEPHALY.** BRISSAUD et BRUANDET, *Nouv. (338) Icon. de la Salpêtrière*, mai-juin 1903, p. 133.

THIS paper contains a full account of the dissection of a case of anencephaly and spina bifida in a seven months' foetus.

The child otherwise was normally formed, but from behind the forehead and extending to the upper part of the sacrum a deep cavity replaced the central nervous system. The lower part of the sacral portion of the vertebral column alone was closed.

The skin stopped abruptly at the margin of the cavity and a thin whitish transparent membrane closed the cavity posteriorly.

The bottom of the cavity was formed by the base of the skull and the bodies of the vertebræ—the normal curvatures, however, being absent.

At the anterior end of the cranial portion of the cavity lay the hypophysis adherent by its base to the bone, its upper end lying free among débris of membranes.

Round about it lay the free terminations of branches of the

internal carotid arteries—small in diameter, ending abruptly and presenting the appearance of ligatured vessels.

A careful dissection of the base of the skull revealed the cranial nerves in their proper position and having their usual distribution. The central ends terminated abruptly a short distance from the foramina of exit.

The ocular vesicles were normally developed and the retina presented its usual layers. The optic nerves were independent inside the skull, there being no chiasma. The 3rd, 4th and 6th cranial nerves were distributed to their respective eye muscles, and the branches of the 7th were followed out on the face. The 8th, 9th, 10th, 11th and 12th cranial nerves were normal.

In the spinal portion of the cavity, the central ends of the spinal nerves were found lying in a groove at each side of the cavity.

Peripherally they were normally distributed and the muscles were healthy.

The spinal ganglia and sympathetic system were present and their structure was normal.

No trace therefore of the central nervous system could be found in spite of the normal distribution of the peripheral nerves.

The question naturally arose as to the origin of these motor nerves which are supposed to require a central nervous system for their development. Nerve cells in the motor area undoubtedly were non-existent.

To account for the presence of the peripheral motor nerves with normal distribution two theories they think may be advanced.

1. That motor cells did exist at an earlier age of the fœtus, and from these the motor nerves developed normally. The cells were destroyed, but the nerves persisted and preserved their normal structure. Or,

2. That the motor cells of the cortex and their peripheral axis cylinders have an independent evolution.

Other cases similar to their own have been recorded in which peripheral motor nerves have been found in the absence of cortical motor cells, and they are inclined to the belief that such cases support the view which assigns to peripheral motor nerves an origin independent of the central motor cells.

Their case, they think, throws no light on the pathology of this condition of anencephaly.

EDWIN MATTHEW.

**THE PATHOLOGICAL ANATOMY OF ACUTE INFANTILE (339) POLIOMYELITIS.** Von Dr. Med. ERNST PRÆTORIUS, *Jahrbuch für Kinderheilkunde*, 58. der dritte Folge, Band 8, S. 175.

THE writer of the paper, after reviewing the various views that have been held with regard to the pathology of acute infantile

paralysis, describes the pathological changes in three cases which he had the opportunity to examine. In the first case the child died when nine months old, some three months after the onset of the disease which had produced paralysis of the left leg.

On microscopical examination there was destruction of the grey matter of the anterior horn of the left side in the lower lumbar and upper sacral segments of the spinal cord. The branches of the central artery were surrounded by numerous granules and round cells. At the seat of the lesion the grey matter of the anterior horns was loaded with granules and round cells.

The antero-lateral tracts of the spinal cord contained many degenerate fibres. The anterior roots at the seat of the lesion were completely atrophied. The posterior horns and posterior columns were normal, though the column of Clarke was involved in one case.

The writer concludes that the pathological conditions found in these cases was undoubtedly due to a myelitis of vascular origin and affected that portion of the grey matter which was supplied by the central artery of the spinal cord.

The involvement of the column of Clarke was another point in favour of the vascular origin of the disease, since the column of Clarke was supplied by a large vessel from the central artery of the cord.

FRED. E. BATTEN.

**CHANGES IN THE SPINAL CORD IN A CASE OF DIPHTHERIAL AND OF ALCOHOLIC PARALYSIS. J. MICHELL CLARKE, *Brit. Med. Journ.*, Sept. 12, 1903.**

THE case of diphtheritic paralysis was in a woman aged 20. There was loss of power in both arms, in both legs, and in the diaphragm. The hands and feet were dropped, and the muscles were wasted, the extensors being more so than the flexors. The R.D. was more or less complete in all the affected muscles. Deep reflexes were absent, but the superfcials active. There were no sensory disturbances except at the very commencement of the illness, when some pain in the hands was complained of. No paralysis of accommodation or of the muscles of the palate or pharynx. The patient died of broncho-pneumonia six weeks from the onset of the paralysis.

On microscopic examination changes were found in the anterior horn cells of the cervical and lumbar enlargements. The anterior mesial group was unaffected, but the lateral groups showed varying degrees of central chromatolysis. Similar changes but of lesser degree were found in the cells of Clarke's column, and in a few cells of the one spinal ganglion examined. In the medulla a

proportion of degenerate cells were to be seen in the vago-accessory nucleus, and in the tenth motor and twelfth nuclei.

With Marchi's stain degenerate fibres were found in the antero-spinal roots, both within and without the cord, also in the vagus and hypoglossal roots. Degenerate fibres were likewise to be seen in the posterior roots, and these could be traced upwards in the posterior columns. Of the peripheral nerves, the anterior and posterior tibials, the musculo-spinal, ulnar and phrenic were examined, and all contained a proportion of degenerate fibres. No fatty changes were found in the heart muscle.

In the second case, which was one of alcoholic origin, the legs only were affected. The changes in the cord were confined to the lumbar enlargement and were somewhat similar to those just described in the diphtheritic case.

W. K. HUNTER.

**ON THE MEDULLATED-FIBRE CONSTITUENTS OF A NORMAL (341) BRAIN AND OF THE BRAIN OF A GENERAL PARALYTIC.** KARL SCHAFFER (Budapest), *Neurolog. Centralbl.*, Sept. 1, 1903, p. 802.

THIS is one of those many contributions which are helping to give us a clear conception of the minute structure—so far as the fibres are concerned—of the cerebral cortex.

A former paper by this author, on the pathological anatomy of the cortex, was criticised by Nissl, the chief burden of the criticism being that Schaffer had failed to check his pathological observations by comparing them with the normal. In pleading not guilty to this crime in the past, he effectually protects himself against a repetition of the accusation in the present case by presenting to us in this paper the normal and pathological conditions (illustrated) side by side.

The results of his investigations into the normal structure of many regions of the cortex are presented in a clear and concise form, and it is interesting to know that they practically agree with Kaes' work on the same subject.

The chief object of his investigations, however, was to determine whether in general paralysis the process of medullated-fibre degeneration in the cortex is a diffuse one, or whether certain regions are exempt from the destructive process. He holds Tuzek to be the founder of the latter view, and to Kaes he attributes the former. In the present paper he describes a case in which the central convolutions, the frontal convolutions of the external surface, the superior parietal lobule, the occipital convolutions, the cuneus, the deep aspect of the first temporal gyrus, the upper half

of the island of Reil, and the cornu ammonis were quite normal, whereas the disease had affected to a marked extent the following portions of cortex: the basal frontal convolutions, the inferior parietal lobule, the frontal pole, the second temporal gyrus, and the inferior half of the insula.

The author states, however, that in another case he finds the whole cortex to be affected, the destructive process being in that case a diffuse one.

He therefore concludes that in general paralysis of the insane the destructive process, as it affects the medullated fibres of the cortex, though often a diffuse one is not always so, as the case which is the theme of this paper goes to show.

J. FROUDE FLASHMAN.

**CONTRIBUTION TO THE STUDY OF THE PATHOLOGICAL  
(342) ANATOMY AND THE PATHOGENY OF CHOREA.**

STANISLAS KOPAZYNSKI, *Rev. Neurol.*, Aug. 15, 1903, p. 751.

HAVING referred to the paucity of published cases of Sydenham's chorea, with autopsy, in which a minute microscopical examination of the nervous system has been described, the writer proceeds to cite the clinical history of a severe case of six weeks' duration, and to give a description of the subsequent examination.

With the exception of hyperæmia of the meninges and slight chromatolytic changes in certain cortical cells, absolutely no morbid conditions were found.

The results of the pathological investigations of chorea by the more recent observers have rather added to than lessened the chaos that exists, the most diverse results being recorded. Among these multiform lesions may be mentioned: hyperæmia and thickening of the meninges, hæmorrhagic pachymeningitis, inflammatory and hæmorrhagic exudations, and areas of softening in the brain, embolism, thrombosis of the longitudinal sinus, chromatolytic and nuclear changes in the nerve cells, and varicosities on their processes.

At the present time two theories as to the pathogeny of chorea have the support of investigators, the one attributing the disease to a diminished cell stability, congenital or acquired; the other claiming an infection by pathogenic organisms to be the principal cause.

Emphasis is laid by the writer, in concluding his paper, on the frequent diminution or absence of the reflexes, especially the patellar, due to the lessening of the muscle tonus.

G. W. HOWLAND.

**THE NEUROCOCCUS OF BRA IN THE BLOOD OF EPILEPTICS.**

(343) TIRELLI and BROSSA, *La Rif. Med.*, No. 34, 1903.

IN 1902 Bra, and in 1903 Bra and Chaussé, published the results of an examination of the blood of epileptics, which suggested a germ origin of epilepsy. They stated that just before, during, and just after an epileptic seizure, an organism could constantly be found in the blood of the patient, and to this organism they gave the name "neurococcus."

They said that it varied somewhat in the forms which it assumed, and that it was sometimes seen as a coccus, sometimes as a diplococcus, and sometimes the cocci grew in chains.

They were slightly refractive, and showed rapid undulatory or rotatory movements.

They grew readily on agar and on potato, in broth and in gelatine.

Injected into rabbits they generally produced convulsions of the tonic type, but sometimes clonic movements were seen. These convulsions showed many of the characteristics of an epileptic seizure.

The animal regained its normal condition in about an hour.

Besta has made a similar examination of the blood of twenty-two epileptics, but failed to find any organisms.

He denies that epilepsy could be attributed to any specific organism.

Quite recently the authors have carried out a similar investigation, and have in some instances found small moving bodies, but they saw no organisms.

They have come to the conclusion that these small bodies are the result of a fragmentation of the morphological elements of the blood, which occurs while the blood is being collected.

R. G. Rows.

**FURTHER CONTRIBUTION UPON THE PATHOLOGY OF**

(344) TETANY, TOGETHER WITH A NOTE ON THE CHEMISTRY OF CALCIFIED CEREBRAL VESSELS (Weiterer Beitrag zur Pathologie der Tetanie, nebst einer Bemerkung zur Chemie Verkalkter Hirngefäße). A. PICK, *Neurol. Centralbl.*, Aug. 16, 1903, S. 754.

THE author has, in a previous communication to the same journal (*Neurol. Centralbl.*, Juli 1, 1902, S. 578), already described two cases of tetany, which on post-mortem examination showed calcification of the minute vessels in the grey matter of the cerebellum and cerebrum, and he again finds a similar condition present in another fatal case of tetany which recently came under his

observation. The patient, a woman of 37, had suffered from tetany for years, and had always been mentally weak. After a confinement in March 1902, her power of vision became increasingly impaired, and in 1903 she was admitted into hospital suffering from "double unripe cataract of tetanoid origin," for which a preliminary iridectomy of the right eye was performed. At a menstrual period which occurred a week after this operation, there supervened a condition of mental aberration and excitement lasting for several weeks, during which she also developed a typical attack of tetany, death occurring fifteen weeks after the onset of these more acute symptoms. In the cerebellum, the author discovered very marked calcification of the vessels in the neighbourhood of the nucleus dentatus, and a similar but much less pronounced condition in the minute vessels of the cerebrum, more especially in the central ganglia.

In certain cases of calcification occurring in the brain, the author and also another observer, v. Gierke, have demonstrated the presence of calcified albuminate of iron, and in the present case the calcified flakes and nodules removed from the vessels in question were found to contain iron (Berlin blue reaction).

The author has now found calcification of the minute vessels in the grey matter of the cerebellum and also, but to a less marked extent, in the grey matter and ganglia of the cerebrum, in four chronic cases of tetany, and he therefore concludes that this phenomenon is a comparatively common if not a constant accompaniment, and may be considered as being not merely an attendant trophic change, but the actual cause of the disease.

W. E. CARNEGIE DICKSON.

**ON THE PATHOLOGY OF TOXIC BRAIN DISEASES.** W. ALTER, (345) *Neurolog. Centralb.*, June 1, 1903, p. 527.

THIS is a paper in which the author seeks, on theoretical grounds, to support the view, now held by so many other workers, that very many cases of mental disease are capable of being explained in terms of toxæmia. He says "the general pathology of the psychoses shows an ever increasing tendency to attribute to autointoxications and to toxic processes in general, a greater rôle in the production of mental disorders." From a statement of Ceni's in the *Neurologisches Centralblatt*, that he could prove the presence of specific autocyto toxins and antiautocyto toxins in the blood of epileptics, Alter has been tempted to show how the various mental affections can be fitted in to the Ehrlich hypothesis. He considers that certain diseases of the nervous system—and especially general paralysis and epilepsy—are due to the union of



specific toxins poured into the blood from some unknown workshop, with preformed substances of the nervous system. As a specially suitable illustration, he takes the case of a general paralytic with many remissions. In this case the toxin unites with the side chains of the nerve cell, and the bad effect on the cell is shown by the advancement of the disease; soon there occurs the over-production of side-chains, which are thrown off into the blood-stream, and there act as the antitoxin, protecting, so long as they last, the nerve cell from the evil influence of the toxin. This period is expressed clinically by the period of remission. Since there is a constant production of toxin, there is a constant repetition of its attacks on the nerve cell which gradually becomes exhausted, and so the disease progresses to its fatal termination.

A similar illustration is given in regard to epilepsy. The author points out the importance of these matters from the point of view of therapeutics, and how very essential it is that investigations should at once be directed towards obtaining an accurate knowledge of the condition of the blood in all cases of mental diseases, and the necessity for combining such investigations with experiments on animals along immunity lines. The results of such methods in other fields of clinical medicine are so encouraging that similar means should not be neglected in the case of the psychoses.

J. FROUDE FLASHMAN.

### CLINICAL NEUROLOGY.

**POLYMYOSITIS IN CHILDHOOD.** Von Dr ARTHUR SCHÜLLER, (346) *Jahrbuch für Kinderheilkunde*, 58. der dritte Folge, Band 8, S. 193.

THE author describes a case of polymyositis in a boy seven years old. At this age, during the course of whooping-cough, the boy developed fever with dyspeptic symptoms and a transient rash. After four days the initial symptoms passed off, and swelling of the eyelids appeared, with painful induration of the muscles of the face and neck. Sensation remained normal and the general condition good, but the rigidity of the muscles extended to the muscles of the thorax, the abdomen and back, and to a lesser degree to the more distal muscles of the upper and lower extremity. During the height of the disease attacks of pain became a prominent symptom, which were especially marked in the forehead and across the shoulders. The acute stage of the disease lasted three weeks, after which the contractions which had been produced became gradually less, and in eight weeks the boy was well.

The diagnosis is fully discussed, and especially with regard to

the invasion of the muscles by trichinae, which the case clinically closely resembled.

No trichinae were found in the stools. There was no eosinophil leucocytosis of the blood, and a radiograph of the limbs showed no calcified trichina capsules in the muscles.

By exclusion the diagnosis of a primary acute polymyositis is adopted.

A very good digest of the various forms of myositis is given, and an extensive bibliography.

FREDERICK E. BATTEN.

**A CASE OF ERB'S JUVENILE DYSTROPHY, ASSOCIATED WITH  
(347) BILATERAL ENLARGEMENT OF THE PAROTID AND  
SUBMAXILLARY GLANDS. J. MICHELL CLARKE, *Brain*,  
vol. 26, 1903, p. 202.**

DR MICHELL CLARKE reports a case of muscular atrophy with enlargement of the parotid and submaxillary glands. The muscular atrophy, though somewhat anomalous, conforms most nearly to Erb's juvenile type.

The onset of both muscular atrophy and swelling of the parotids was simultaneous, and came on without any definite cause and without pain. The case is that of a boy aged 18, the eldest of a family of five, all of whom are healthy. The boy was healthy till 13 years old, when the first symptoms, viz., weakness of the legs, was noticed. About the same time, swelling of the parotids gradually developed, and, having attained a certain size, remained stationary. The boy was able to walk, but could not go upstairs. He was unable to rise from the floor when placed on his back.

The muscular atrophy in the trunk and limbs was very pronounced and affected the proximal muscles of the limbs rather more than the distal. The serratus magnus was small but its action was normal. The lower two-thirds of each rectus abdominis was more affected than the upper third. On electrical stimulation the affected muscles show a diminished response in proportion to the degree of atrophy present. There was, however, no qualitative change.

Tendon reflexes were all absent. The plantar reflex was difficult to obtain, and generally of flexor type.

The arrested growth had affected all parts of the body, and the small size of the bones exemplified the general arrest of growth which seems to have been coincident with the onset of muscular atrophy. Intelligence was good.

FREDERICK E. BATTEN.

**NOTE ON "TOXIC DEGENERATION OF THE LOWER NEURONES  
(348) SIMULATING PERIPHERAL NEURITIS." R. T. WILLIAMSON, *Brain*, Summer 1903, p. 206.**

UNDER the above title, the reviewer has described (*Brain*, 1902) a series of cases with the post-mortem results of one case, and Dr Williamson brings forward another case of the same kind which came under his care in 1899. This patient was a woman, forty years of age, who in June 1899 suffered from an acute illness, the chief symptoms being pain over the limbs and trunk. She was very ill for eight days and then slowly recovered, but for three weeks she felt unwell. She then returned to her work and continued well till August 1899, when a second attack of a similar nature occurred, and was thought by the patient to be influenza. After a few days the acute symptoms subsided, leaving her very weak. About ten days after the pains had disappeared, she noticed that the hands were getting feeble. The weakness gradually spread, but there was no pain numbness nor tenderness. Nothing in the history or condition of the patient pointed to diphtheria, lead or alcohol.

On admission to hospital (October 1899), there was marked atrophy of the thenar and hypothenar eminences and of the interosseal muscles of each hand. There was paralysis of the extensors of the wrist and fingers. The upper arm, trunk and lower extremity muscles were unaffected. There were no cranial or sensory changes, and the knee-jerks were normal.

Gradual improvement occurred, and by Feb. 1901, she had completely recovered. No relapse had occurred in March 1903.

The author refers to the difficulty of giving any name to the condition, which clearly was not anterior polio-myelitis and was not the ordinary form of peripheral neuritis. He concludes that the case belongs to the group which had been described by the reviewer under the title of "toxic degeneration of the lower neurones."

STANLEY BARNES.

**THE SYMPTOMS OF LESIONS OF THE UPPER PART OF THE  
(349) PONS. Professor RAYMOND and M. R. CESTAN, *Gaz. des Hopitaux*, July 18, 1903.**

THE authors draw attention afresh to a symptom-complex which they have previously dealt with in another paper, and which they regard as diagnostic of lesions commencing in a particular region of the pons. The symptoms are seen in their characteristic form

only in the early period of the clinical history. They are as follows.

When the patient is looking before him his face has its normal aspect; but when he attempts to look to either side it is observed that the eyeballs are unable to reach the outer canthus, and lateral nystagmus occurs during the attempt. This paresis is more marked on looking to one side than to the other. The movement of convergence can be carried out in the normal way, and without nystagmus. The up-and-down movements of the globes are also unimpaired, but they are associated with some nystagmus. There is no ptosis. The pupils react to light and accommodation. So far, then, the syndrome differs from that of Millard-Gubler, since the eyeballs at rest occupy the median position without internal strabismus, showing that the nucleus and fibres of emergence of the sixth are intact; and from that of Weber, in that there is no affection of branches of the third pair.

Associated with these ocular symptoms is a hemiplegia of arm and leg. The face may also be slightly involved, but often escapes for a time. In the affected arm and leg there is no great loss of motor power, but there is tremor, which is increased by voluntary movement; there is inco-ordination, which is increased on closing the eyes; and there are also athetoid movements of the fingers. The reflexes are exaggerated. The plantar reflex may be extensor or flexor. Sensory symptoms are also present in the form of formication and other subjective disturbances, along with diminution of all forms of sensation, and more or less complete disappearance of the stereognostic sense.

At a later period the characteristic clinical picture is altered by the supervention of other symptoms, such as internal strabismus or facial paralysis.

In three cases presenting these symptoms the authors have made a post-mortem examination, and in each they have found a lesion with a special localisation which corresponds to the early stage of the symptoms. The position in which the lesion starts is the tegmental region of the upper part of the pons. The lesion lies between the nuclei of the third and the sixth nerves, neither nucleus being involved. A lesion in this position can involve the sensory and the cerebellar paths, while the motor paths are spared. Histological examination confirmed the integrity of the sixth nerve and nucleus, and also of the nucleus and fibres of the third and fourth nerves. The ocular paresis, therefore, must have an extra-nuclear origin. In short, the lesion, situated between the third and sixth nuclei, involves the fibres which pass from the sixth nucleus to that part of the third which supplies the internal rectus of the opposite side.

W. B. DRUMMOND.

**CHRONIC PROGRESSIVE HEMIPLEGIA, WITH REMARKS ON  
(350) TWO CASES OF UNILATERAL PARALYSIS AGITANS  
WITHOUT TREMOR.** HUGH T. PATRICK, *Journ. of Nerv. and  
Ment Dis.*, Aug. 1903.

DR PATRICK, in this paper, describes with but little comment, three very interesting cases of affection of the right side of the body.

The first is that of a girl who had been healthy until the age of fourteen, when she noticed weakness and clumsiness of the right hand and foot, which slowly and steadily became worse. When seen at the age of eighteen she presented slight right facial weakness affecting the upper and lower segments equally, and pronounced weakness of the muscles supplied by the fifth nerve. There was no affection of the tongue nor of the other cranial nerves. The right arm and leg were weak in all movements proportionately, and the muscles of this side were smaller than those of the left. Sensation was normal in all respects when tested, but the patient noticed something different between the two sides, and the right limbs occasionally felt numb. The reflexes were exaggerated, and there was a suggestion of ankle clonus; the plantar response was of extensor type. In spite of this the limbs appeared flaccid, and the gait was not spastic or ataxic, but of moderate steppage character. The only abnormality discovered in the other systems was a slight enlargement of the right side of the thyroid. *Faute de mieux* Dr Patrick is inclined to consider the case as "a slowly increasing degeneration of the pyramidal fasciculi or of the cerebral motor neurone system."

The other two cases are much alike, and are described, probably correctly, as cases of unilateral paralysis agitans without tremor. The only marked difference between them was that, in the first, the facies was normal, whereas, in the second, it was typically Parkinsonian. Both suffered from stiffness and rigidity of the right arm and leg with but little loss of actual power, and both complained of a dull heavy aching in the muscles. In both the hand was habitually held in the typical Parkinson position, and on exertion there was a tendency for the right arm to adopt the typical attitude with sometimes slight but typical tremor. The deep reflexes in both were more marked on the affected side, and in both the plantar reflexes were of flexor type. In the first case there was a marked difference between the size of the muscles on the two sides of the body, those on the right being the smaller. This was true of the second case, but to a much less extent.

H. DOUGLAS SINGER.

**MYOCLONUS MULTIPLEX AND THE MYOCLONIAS; REPORT (351) OF CASES AND AN ATTEMPT AT CLASSIFICATION.**

CHARLES L. DANA, *Journ. of Nerv. and Ment. Dis.*, August 1903.

THIS paper aims at a classification of the various forms of disease in which myoclonus, or non-rhythmical spasmodic contractions of muscles, is the prominent symptom. Details of three cases and summaries of five others are given, illustrating four of the groups. The classification is as follows:—

I. Myoclonia of Friedreich, or peripheral type, including Myokimia. Paramyoclonus Multiplex of Friedreich; astasic myoclonia of Vanlair; multiple spinal myoclonus of Lowenfeld; fibrillary chorea of Morvan; and fibrillary myoclonus of Kny.

II. Myoclonia of the functional or hysterical type. Chorea major; and the chorea electrica of Henoch (*this is more generally recognised as a form of Tic.*)

III. Myoclonia of the Convulsive Tic type. Myospasms; memory-spasms of Friedreich; habit chorea; chorea variable des dégénérés; convulsive or spasmodic tic; tic general; Tourette's disease; myriadrit (*more correctly myriachit*); palmus; tic neurosis of Collins.

IV. Myoclonia of the degenerative chorea or "familial" or myoclonus-epilepsy type. Degenerative chorea; hereditary chorea; Huntington's chorea; Unverricht's familial myoclonus; myoclonia congenita of Seeligmüller(?); hereditary degenerative chorea of Sachs.

V. Myoclonia of the type of the infectious and symptomatic choreas. Chorea minor; Sydenham's chorea; chorea electrica of Dubini(?); chorea electrica of Bergeron; senile chorea of Gowers(?).

In the above the queries are by Dr Dana, but the italics by the present writer.

Of the illustrative cases the first closely resembled that described by Friedreich as Paramyoclonus Multiplex (a translation of Friedreich's paper is given as an appendix), but presented in addition marked Myokimia, a fact which the author considers of importance as showing the close relation between myokimia and the severer types of bundle clonus to which the term paramyoclonus multiplex, if used at all, should be restricted. The second case had suffered for thirty-one years from seizures recurring at irregular intervals in which there were rhythmical movements affecting the face, neck, shoulder and arm muscles, together with fibrillary tremor, irregular clonic movements, and tonic contractions with and without locomotor effect. Case 3 presented spasms affecting in the main co-ordinate groups of muscles, causing

grimaces, wry-neck, and movements of the shoulders and trunk. There were also, although much less prominent, isolated muscle spasms without locomotor effect, fibrillary and fascicular spasm. The remaining five cases are examples of degenerative chorea and tic, all following attacks of Sydenham's chorea.

H. DOUGLAS SINGER.

**ALCOHOLISM IN BRITTANY.** A. DUCREST DE VILLENEUVE, (352) *Nouvelle Iconographie de la Salpêtrière*, No. 3, May-June 1903.

THE author of this article is of opinion that in the peasants of Brittany, notwithstanding their apparent calmness of disposition, one is dealing with a race of nervous temperament. Several physicians have referred to the considerable number of hysterical patients met with in Brittany, and the author looks on the prevalent alcoholism as a factor in the production of this state of matters. He deals only with the rural population. Without being inveterate drinkers the peasants of Brittany "do nothing by halves," and drink their *eau de vie* out of large glasses. Cider is the national beverage in name only.

Apart from the relatively large number of public-houses in the villages, a special cause of the evil is to be found in habits in drinking healths, feasts, etc., which may in part be traced to ancient Gallic or Druidical sources. Thus in dealing with the problem, and endeavouring to diminish temptations, one has to reckon with what is deeply planted in the race.

A. HILL BUCHAN.

**INVESTIGATIONS CONCERNING "VIBRATIONSGEFÜHL," OR (353) THE SO-CALLED BONE SENSIBILITY (PALLÆSTHESIA).** RYDEL und SEIFFER, *Arch. f. Psychiat. u. Nervenk.*, Bd. 37, H. 2, 1903, S. 488.

TREITEL in 1897 and Egger in 1899 published the results of their investigations on the nature and significance of "vibration-sensations." By this is meant the sensation one experiences when a tuning-fork in vibration is placed on some subcutaneous bony surface, *e.g.* that of the tibia. It is to be noted (for a reason stated below) that the term "bone-sensibility" (*knochensensibilität*, *sensibilité osseuse*) is misleading, and the authors of this paper prefer to say "sense of vibration." Comparatively little is known of the subject outside of France, so that a scientific contribution to it is to be welcomed.

The authors examined the phenomena in fifty or sixty nervous cases, employing for the purpose a tuning-fork 23 cm. long, weighing 100 g., and vibrating sixty-four times per second. The addition of movable clamps to the arms of the tuning-fork enabled them to vary the vibration time of the instrument, while a very ingenious device (for details see the original) of an Italian otologist, Gradenigo (adapted to one of these clamps) allowed the accurate (relative) measurement of the duration of the vibration-sensation.

The condition in health was first carefully tested, no less than fifty-four points on the body, from the middle of the cranium to the sole of the foot, being systematically examined. Great importance was attached to the *duration* of the sensation when the tuning-fork was placed on a given point, and this was used as a standard of comparison for the different parts of the body. For instance, the sensation is felt for a longer time at peripheral points than at proximal; the duration is likewise longer if there be muscle rather than bone beneath the part on which the instrument is vibrating, and proportionately longer at the ends of bones than in the middle.

Under pathological conditions some interesting results were obtained. In peripheral neuritis and in multiple sclerosis no definite rule could be laid down; it was found, however, that there was practically always very considerable impairment of this new "sense," though there was very little alteration in the ordinary cutaneous sensations. In syringomyelia there was always a fairly definite correspondence between the affected skin areas and the loss of vibration-sensation.

In twenty-two cases of tabes the general conclusion is come to that there is a close connection between the ataxia and the impairment of this sensation, and this is well illustrated by charts. There seems also to be some evidence for the view advanced by the authors that the sense of position in space is similarly related to "pallæsthesia" (παλλω, to vibrate, whirl), the name which it is proposed should be given to the vibration-sensation.

In Friedreich's ataxia, contrary to the usual condition, the ends of long bones seem to be less sensitive than the middle. The authors are of opinion that the sensation does not depend on afferent nerves in bone, because the sensation is experienced although the tuning-fork is placed on some part where there is no bone. Similarly, it does not appear to be either periosteal or muscular in origin. They suggest that it arises in the finest nerve arborisations lying just under the skin, and that it is akin to sensations which come from muscle tendons and fasciæ, joints and joint-capsules.

S. A. K. WILSON.



**THE PLANTAR REFLEX OF INFANTS.** The Diagnostic and (354) Prognostic Significance of Extension and of Flexion of the Toes. ANDRÉ LÉRI, *Rev. Neurol.*, July 30, 1903, p. 689.

AFTER a systematic research upon 166 children, the author has arrived at the following conclusions:—

(1) At the time of birth an extensor response is obtained. A flexor response is met with only in very rare instances.

(2) After the third year a flexor response obtains. An extensor reflex is exceptional, and when present it has not such a certain diagnostic value as it has in the adult.

(3) Between the first and the third year the extensor response is only occasionally met with in healthy children. The extensor reflex is often met with during this period of life under the following conditions:—

(a) In affections of the central nervous system.

(b) Where profound disturbances of general nutrition exist.

(4) The type of the plantar reflex usually changes from extensor to flexor at the fifth or sixth month. The change may take place in one foot some time before it appears in the other foot.

(5) The extensor reflex seems to disappear synchronously with the disappearance of the spastic attitude of the newly born.

(6) The presence of a flexor plantar reflex may be of great prognostic value in children who, prematurely born or ill-developed from some cause, are late in learning to walk. In such children the presence of a flexor response enables one with certainty to give a good prognosis as regards the ultimate power of walking, and is a sure indication that the symptoms of spastic cerebral diplegia will not appear subsequently.

JAMES COLLIER.

**CONCERNING THE CREMASTERIC REFLEX AND VOLUNTARY**  
(355) **CONTRACTION OF THE CREMASTER MUSCLE.** G. PERUSINI, *Rivista di patologia nervosa e mentale*, 1903, p. 318.

THE case here described is that of a man of 29—alcoholic, epileptic, and a sexual pervert—who was for a time under observation in the asylum of S. Maria della Pietà in Rome. The patient showed no obvious physical abnormality save the one to be presently described with regard to the reflexes. The pupils reacted normally. The conjunctival reflex was sluggish, the pharyngeal reflex abolished. The tendon and periosteal reflexes of the extremities were brisk. The axillary, epigastric, popliteal and plantar reflexes were faint. The abdominal and gluteal reflexes, on the other hand, were brisk. The scrotal reflex, on stimulation by cold, was absent. The peculiar point in the case, however, was the cremasteric reflex, which was

not only elicited by the ordinary methods of stroking the inner surface of the thigh, or on pressure some distance above the internal condyle, but also appeared when the patient clenched the fist or strongly flexed the knee. The phenomenon did not occur if the extensor muscles came into action together with the flexors of these joints. Finally it was observed that while the patient's abdominal muscles remained flaccid—a fact confirmed by palpation with the hand—he could voluntarily and repeatedly draw up one or other testicle at will independently on the two sides, the testicle being pulled up to the inguinal ring and held there. The dartos muscle, as already mentioned, took no part in the phenomenon.

The cremasteric reflex is almost constantly present in health. It may be abolished in certain local affections, *e.g.* varicocele and, as Gibson of Edinburgh has pointed out, in some cases of sciatica. In organic hemiplegia it is not infrequently abolished on the affected side. In functional nervous diseases its behaviour is variable, being sometimes diminished or lost, sometimes exaggerated. Feré in 1899, and Mingazzini in 1901, each recorded a case in which synergic contraction of the cremaster occurred when other muscles, *e.g.* of the hand, were thrown into action. Perusini's case is the first in which an isolated voluntary contraction of the muscle has been observed.

PURVES STEWART.

**ADDUCTOR REFLEX OF THE FOOT.** R. HUSCHBERG, *Rev.* (356) *Neurol.*, Aug. 15, 1903, p. 751.

A REFLEX movement of adduction of the foot may be produced by stroking the side of the foot, especially in the region of the great toe.

This is only present under pathological conditions, and is always present when Babinski's reflex is positive, and it may therefore prove of value when the extensor response is obtained with difficulty, or is absent.

G. W. HOWLAND.

## PSYCHIATRY.

**ORGANIC PSYCHOPATHIES (PSYCHOPATHIES ORGANIQUES).** (357) E. DUPRÉ (of Paris), 382 pp. Extract from the *Traité de Pathologie Mentale* of Ballet, 1903.

MEDICAL literature swarms with references to the psychic disturbances in organic lesions of the nervous system, but no one as yet has published such a systematic analysis of these disturbances as Dupré in his Organic Psychopathies. As the lesions embrace the whole of cerebral pathology and the psychic disturbances of all the elementary forms of mental alienation, the task of correlation is

not easy. As the characteristic of the work is fine clinical observation and subtle psychological analysis a resumé is quite inadequate. The first half of the book is devoted to general paralysis of the insane. D. insists on the demential nature of the disease at its very outset: in the second stage this demence becomes "global," but the symptoms merely become exaggerated without change of form: psychic synthesis has become impossible, there is merely juxtaposition of isolated concepts and not combinations of associated ideas.

An early symptom very often is "parectropie," by which D. means a difficulty in the voluntary execution of movements commanded, especially in the face: this disturbance in the cortical psycho-motor association is also present in the majority of dementias.

Among the symptoms of the second stage are the troubles of speech and language: the latter are very minutely analysed. As the troubles of speech are Dyslogia, Dysphasia and Dysarthry, so we have psychographic troubles of great pathognomonic value depending on the mental disturbance, calligraphic troubles depending on neuromuscular ataxia and paresis, and rarely Dysgraphia or Agraphia corresponding to the Dysphasia.

The diagnosis of G. P. in the first stage from neurasthenia may be very difficult, as one can have the association of neurasthenia and G. P., G. P. simulating neurasthenia, and a pseudo-paralytic neurasthenia in a patient who has previously had syphilis. D. insists that neurasthenia is a mere syndrome, and suggests that grave neurasthenias may be the beginning of G. P. but capable of arrest, temporary or definite. Syphilitics under treatment may present a hystero-neurasthenia simulating early G. P. The delirious troubles in G. P. are distinguished from maniacal and melancholic states by an analysis of the intellectual content and moral tendencies of the paralytic: the ideas show varying character and absurdity, and his phantoms do not enter the metaphysical world.

In the second stage the difficulty is to diagnose G. P. from alcoholism, saturnism, Korsakow's syndrome, acute mental confusion of toxic origin, cerebral syphilis. The mental state of the alcoholic is different, the character and intelligence both reacting differently: memory shows gaps but is not abolished. Treatment helps the diagnosis as also from saturnism, where it may be impossible to analyse the polyneuropathic complex of a hystero-neurasthenia on a basis of intoxication.

In the final stage one has to diagnose from the organic dementias.

Lymphocytosis of the cerebro-spinal fluid is one of the most precocious and constant symptoms: D. holds that it is occasionally absent.

Etiologically G. P. is the result of hereditary predisposition and toxic processes due usually to syphilis, alcohol and overwork: pathologically it is a diffuse encephalo-meningitis of subacute nature, neither primarily interstitial nor primarily parenchymatous, but the reaction to the paratoxic cause in general. D. sees the fundamental macroscopic character of the cerebral lesions in the adherences of the pia to the cortex: as a matter of fact this lesion is only found in a minority of cases. As to mercurial treatment D. "has never seen the benefit, sometimes observed its harmlessness, often its drawbacks and dangers."

In the second chapter D. deals with the Psychic Troubles in the Organic Encephalopathies: he divides the lesions into circumscribed, multiple, diffuse.

As to circumscribed lesions, psychic symptoms are usually general, not localising symptoms, and among the earliest "psychicity is in the series of cortical functions a reagent much superior to motricity and sensibility."

The psychic state after localised hæmorrhage or softening with the strange gaps of systems in memory and conscience is fully described. In discussing the disorders of speech, D. insists on the importance of the distinction between articulation and intonation. He criticises the antiquated schemes of explanation depending upon superior psychic centres which are mere "anatomical phantoms endowed by us with a superior functional activity, but which vanish at the first attempt at objective localisation in the brain." Psychology equally disavows the creation of these superior centres where is elaborated an abstract psychic activity, "a quintessence of the mental personality."

After discussing the associated troubles—agnosis, psychic blindness, psychic deafness, astereognosis—D. analyses the disorders of psychoreflex action and of the mimic faculty. As regards facial mimic the cortex is the centre of psychic elaboration, the thalamus of the automatic organisation, the bulb of the neuromuscular execution of the psychoreflex process of emotive expression. Various affections show pathognomonic masks.

With regard to tumours, D. holds that the tolerant ("silent") tumours from the somatic point of view are intolerant from the psychological. Intoxication of the cerebral substance by cellular poisons of neoplastic origin may account for psychic symptoms in cerebral tumours and abscesses.

With regard to the chronic Sclerotic Encephalopathies of the adult, D. agrees with Marie that in tabes intellectual changes are rare, moral are frequent. Tabetic inability to walk is often due to a phobia associated with the tabes—it is a psychic trouble of emotional origin but intellectual content. This stasobasophobia is not to be confused with the astasiabasia of the hysterics. In

multiple sclerosis weakening of the intellect is common ; in the terminal stage of syringomyelia psychic disturbances occur. In the various diplegias the mental state is also various.

After a review of the tubercular, syphilitic and vascular psychopathies D. analyses the mental condition in the organic dementias: from the latter he deduces the preponderance of the intellectual rôle of the left hemisphere.

C. MACFIE CAMPBELL.

**THE IDEO-OBSESSIVE CONSTITUTION.** Dr SERGE SOUKHANOFF, (358) *Revue Neurol.*, juin 30, 1903, p. 613.

CASES of the type described by Soukhanoff in this interesting paper are easily recognisable and spring instantly to memory. The individuals of the category are over scrupulous, disquieted over trifles, indecisive in action, and anxious about their affairs. They are given early to morbid introspection, and are easily worried about their own indisposition or the illnesses of their friends. They are impressionable, possess considerable *amour propre*, and are easily offended; but as they are usually reticent they hide their resentment, and are often secretly ashamed of those bizarreries peculiar to their disposition. They are often timorous and apprehensive and prone to pedantism. The moral sentiments are pronounced in most cases, and if they are, as a rule, somewhat exigent and egoistic, they have a lively sense of their own defects.

These characteristic traits may not, of course, be present in all cases, or some may preponderate at the expense of others, but what to Soukhanoff seems certain is, that in the great majority of cases an *ensemble* is evident which justifies their inclusion under a category as ideo-obsessive. This ideo-obsessive state is, according to the author, amongst the congenital anomalies of the neuro-psychic organisation, and may be considered as a form of morbid autonomy.

On this scrupulo-inquiet foundation described above they develop dominant ideas, fears and hallucinations early in life. The manifestations are thus multiple and protean in form (*folie du doute, le délire du toucher, l'agoraphobie*, etc.), but these are but divers phenomena of one fundamental malady.

The course is fluctuating, and is affected amongst other things by puberty, involution of the organism, the moral emotions, and somatic illnesses. A temporary melancholia may supervene, and the obsessive state become more marked, giving the clinical picture of melancholia with obsessions, but in cases of this kind it can be shown that prior to the melancholia the patient was of this type. This character obtains during the life of the individual, and though the obsessions may not be very marked, the fundamental character and bias persist.

In affirming this definite ideo-obsessive character, the existence of isolated obsessive ideas, fears, etc., is not negated, as compounds of other clinical syntheses. Observation, indeed, demonstrates that obsessions may be encountered as episodes in hysteria, dementia præcox, general paralysis, etc., and their occurrence in no way, says Soukhanoff, invalidates their inclusion under a separate class of cases, ideo-obsessive in type, any more than an episodic melancholia occurring in general paralysis would contradict its inclusion within the latter group.

Notwithstanding its prolonged evolution this state rarely terminates in marked mental enfeeblement, though in some cases there may be developed symptoms relevant to central arterio-sclerosis.

In certain cases the symptoms are so marked as to resemble a psychosis rather than a psychopathy, and Soukhanoff is inclined to believe in transition cases from ideo-obsession to delirium. Soukhanoff's cases have shown clear recollections of their past life, particularly for painful or sad events or tales impressed in early childhood, and he thinks that these may play a certain rôle in their subsequent development of obsessive ideas and fears.

Finally, the author states that these patients are frequently descended from parents with a similar mental constitution, and that often this is inherited by the son from the mother and by the daughter from the father. A certain frequency of association of the state and pulmonary consumption is also noted, and is of suggestive interest.

R. CUNYNGHAM BROWN.

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# **Review**

of

# **Neurology and Psychiatry**

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## **Original Articles**

### **A NEW METHOD OF STAINING NERVE CELLS (STAINING AND DIFFERENTIATION IN BULK).**

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(The most important step in the process about to be described, differentiation with a mixture of alcohol and ether, was suggested by Dr Louis Jenner, to whom the writer's indebtedness and best thanks are here expressed for his kind permission both to make use of the suggestion and to publish this note.)

THE methods for the differential staining of nerve cells which are principally employed at the present time—those of Nissl, of Held, and of Hoyer—are beset with drawbacks. The preparation of serial sections by these methods is laborious, and it is often difficult to obtain good and flat sections, owing to the effect of the reagents employed upon the tissues. Constant and reliable results necessitate most skilful technique and a long experience, since over-differentiation and under-differentiation are possible. When aniline oil is used as the differentiating agent, it is almost impossible to prevent subsequent fading of the stain. Moreover, the vapour of aniline and cajeput oil have a most irritating effect upon the mucous membranes of some people.

The method here described has been exclusively used for the past two years in the pathological laboratory of the National Hospital, and it is claimed to have the following advantages:—

1. Simplicity.
2. Rapidity.
3. Perfect serial sections are easily obtained.
4. There is no danger of over-differentiation.
5. The results are constant, and the pictures produced differ in no respect from those produced by the method of Nissl and by Hoyer's thionin method.
6. Fading of the coloration does not occur so soon or so readily as with the above-mentioned methods. If kept in the dark, preparations are still perfect after two years.

#### PROCEDURE.

*Hardening.*—After preliminary hardening in formalin 5-10 per cent. for four days, pieces 4-6 mm. thick are placed in absolute alcohol, which should be changed after four hours for a larger bulk of absolute alcohol. The pieces should remain in absolute alcohol at least seven days, and may with advantage be left several weeks. Perfect results may be obtained with material which has been for twelve months in absolute alcohol. Prolonged immersion in formalin is harmful in proportion to the acidity which the formalin solution has acquired, and according to the duration of the immersion. Old formalin-hardened material should be treated for at least a week in several changes of absolute alcohol before section.

*Staining.*—The pieces are transferred from absolute alcohol to the stain, and are left for one hour at a temperature of 50° C. (the bottle containing the pieces while staining is placed upon the top of the paraffin bath).

*Stain.*—Methylene blue, patent B. 2 grammes; distilled water, 100 c.c. This solution may be used over and over again.

*Differentiation.*—The tissues are transferred direct from the stain into the following fluid:—Absolute alcohol, 90 pts.; methylated sulphuric ether, Sp. G. .730, 10 pts.

This mixture must be freshly prepared, and the proportions kept constant during the differentiation by carrying out the process in stoppered bottles or closely-covered capsules, to prevent the evaporation of the ether.

The alcohol ether mixture is changed as soon as it becomes

deeply coloured, and renewed. It should be renewed every five minutes for twenty minutes, and the pieces subsequently placed in a larger quantity of the mixture for one hour. By this procedure both differentiation and dehydration are obtained.

The pieces are transferred direct to chloroform, which should be quickly changed twice.

After remaining an hour in chloroform they are placed in a paraffin bath (two changes) for not more than thirty minutes; they are subsequently embedded and cut.

The sections are floated out on warm tap-water and fixed to the cover-glasses by the dessication method.

The paraffin is dissolved away with xylol, the sections are blotted with hard filter paper, and mounted in thick turpentine colophonium. It is convenient to render the mounting medium more fluid by standing the bottle containing it in hot water during use.

If after the removal of the paraffin from the sections the latter appear too deeply coloured, they should be dipped for a few seconds in the alcohol ether mixture and returned to xylol before mounting.

This process is applicable also to the staining of individual sections prepared either by the paraffin or the celloidin methods, and to sections cut without imbedding. If celloidin sections are used, the celloidin must be completely removed by washing in a mixture of absolute alcohol and ether in equal parts. After the removal of the paraffin or of the celloidin, the sections are placed in absolute alcohol for at least twenty-four hours. They are stained for five minutes in the methylene blue solution at a temperature not exceeding 50° C., transferred direct to the differentiating fluid, cleared in xylol, and mounted.

The following points should be borne in mind when using this method :—

1. Methylated alcohol should never be employed.
2. The methylene blue should be that supplied by Grubler under the mark Patent B.x.
3. The proportions of alcohol and ether in the differentiating mixture should be constant.
4. Prolonged immersion of the tissues and sections in absolute alcohol, previous to staining, is desirable.
5. A rapidly drying medium should be used for mounting.

Balsam is not satisfactory, and if acid, its use is followed by rapid fading of the stain.

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### **HÆMORRHAGE INTO THE SPINAL CORD (HÆMATOMYELIA).**

By WILLIAM ELDER, M.D., F.R.C.P.Ed.,

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THE following case, which I believe to have been one of hæmorrhage into the spinal cord in the lower cervical region, presents some features of unusual interest. The case was one where the diagnosis was made from the symptoms soon after his admission to hospital, but the further progress of the case has not led me to alter the diagnosis, and, as the patient is still alive and improving, the necessary definite proof of the existence of the hæmorrhage is lacking.

Cases of hæmorrhage into the vertebral canal may have various seats. It may either be outside the cord or into the cord. If outside the cord it may be extra-dural or intra-dural. The commonest cause is traumatism, such as fractures, dislocations or blows, but it is believed to result from cold and exposure, and may be secondary to other affections, such as myelitis, syringomyelia, tumours of the cord or its surroundings, or it may occur in cases of tetanus or convulsions or in acute infectious diseases (Osler).

The most common situation for hæmorrhage, either into the cord or outside it, is in the lower cervical and upper dorsal region where the vertebral column is most exposed to traumatism, and it is in that region that I believe the hæmorrhage occurred in this case.

*Case.*—The patient, a young man aged 22, was admitted to Leith Hospital on 25th September 1902. He was brought in by the Police, by whom he had been found in the evening unconscious in one of the dock sheds. He had a strong odour of alcohol and it was thought necessary to wash out his stomach, after which he recovered consciousness. He stated that he had had a considerable amount of alcohol that morning, although he was not in the habit of taking drink. He remembered having


dinner about 2 P.M., but very little afterwards, and could give no account of how he got to the dock shed, where the Police say he was from about 2 to 8 P.M.

Next morning he did not feel well, and was unable to be discharged from hospital. He complained of pains all over his body of a stinging and shooting character. He also had a numb feeling in both hands, and he had lost much of his power of gripping. His previous history was ascertained to be good. He had never been thoroughly intoxicated before, although he took a pint of beer occasionally. When ten or twelve years of age he had had inflammation of the lungs. There was no history of specific disease. His family history was good, but it is interesting to note that his younger brother was admitted to Leith Hospital a few months later suffering from tremors of his hands, for which he was dismissed from the navy. These tremors were of the nature of the tremors in paralysis agitans. Examination of the nervous system of the patient soon after admission showed the following condition:—He was bright and intelligent after the effects of the alcohol passed off. Hearing and sight were normal. Pupil reactions were normal. Slight nystagmus when eyes turned to the left.

*Sensory Functions.*—There were feelings of numbness and tingling in both hands; the pains all over the body noted above soon passed away. The sense of touch all over the body was normal. His sense of pain was well marked in the trunk, legs, and greater part of the arms, but in a region over the back of the hand and middle, ring and little fingers on the right side the sense of pain was almost entirely lost, whilst the sense of pain was impaired over the dorsal surface of the forefinger of that hand. In the left hand the sense of pain was impaired over the dorsal surface of the four fingers, whilst a band of complete loss of sense of pain stretched across the dorsal aspect of the hand a little below the wrist, leaving, however, the thumb normal.

The power of distinguishing heat and cold was lost over the same region as the pain sense, but the rest of the body was normal as regards the temperature sense.

He had thus, over the dorsal aspect of both hands lost or impaired sense of pain and temperature, whilst the sense of touch was normal. (Dissociation Symptoms.)



He had pains on pressure over the sixth cervical vertebra, and he had a feeling of dull pain or heaviness over the lower cervical region.

*Motor Functions.*—Right arm.—The power of extension of the right elbow was much weakened, almost entirely lost, whilst the power of flexion was fairly good. At the right wrist the flexor power was very weak, whilst the extensor, although weakened, was much stronger than the flexor. In the left arm the symptoms were almost the same as in the right, but there was much less power in the hand, the power of extension being as weak as that of flexion. In this arm also, although the power of extension at the elbow was almost gone, flexion remained fairly good.

The power of movement in his legs when lying in bed seemed normal, but he could not walk because of difficulty of holding himself erect and from an increase in the pain in the lower cervical region.

*Reflexes.*—The supinator jerk could be elicited in both arms—that of the triceps was present, but slight, on the left side, whilst it could not be elicited on the right. A biceps reflex was got in both arms. The patellar reflex was exaggerated on both sides. Both knee and ankle clonus could be induced on both sides, whilst the tendo-Achilles jerk could be elicited in both limbs. Babinski's sign was present on both sides. Cremasteric reflex was absent, whilst epigastric was present on both sides.

Organic reflexes were normal. It was impossible to test his powers of co-ordination on account of the muscular weakness of his arms and hands, and it was not thought judicious to ask him to stand or walk. His alimentary and respiratory systems were normal. His urine contained no albumen and no sugar. As regards his circulatory system, it was normal when he lay in bed, but it was noticed a few days after his admission that whenever he raised his head or the upper part of his body to a sitting position he became pale in the face, felt faint, and his heart's action increased very much in rate. This symptom continued for some weeks, and it was believed to be due to implication of some of the nerve fibres whose function it is to innervate the heart. Patient remained in much the same condition for about a week, when it was noted that the power of flexion of the right hand was improving; the sensory dissociation



symptoms, however, continued. The patient slowly but gradually improved, the power in both arms and hands returning gradually; the muscles chiefly affected showed symptoms of partial atrophy, such as those of the thumb and inter-ossei, but as the power increased this atrophy became again less evident.

The power in his legs, although pretty strong, was not so great as it should have been, and very soon the reflexes had become more marked, so that a gentle tap on the patellar tendon was sufficient to set up a distinct clonus in the whole limb. This was a little more marked on the left than on the right side.

The sensory dissociation symptoms continued for a few weeks, then they became less marked, the right side improving more rapidly than the left, and the sense of pain returning earlier than the sense of temperature.

On November 2nd, about six weeks after admission, it was noted that the power of grasping as tested by the dynamometer was stronger in the right than in the left hand, but both flexor and extensor power had returned to both hands, although weak. He had regained his power at both elbows. He was able to walk without dragging the left leg, as had been noted on his getting up first. His gait was very slightly spastic in character. His sense of touch was normal, pain also was now normal, but the sense of temperature was still impaired over the dorsal aspect of the fingers of his left hand. He also still felt some tingling in the fingers of that hand. When up he complained of a feeling of stiffness in the back of his neck, which disappeared when he lay down.

The patient was discharged in much the same condition. A short time after his discharge he was admitted to the Royal Infirmary, Edinburgh, where he continued in much the same condition, except that the dissociation symptoms disappeared and the spastic condition of his legs became more marked. The power of his hands still remained below normal, and there was also some evidence of atrophy of the muscles of the hands.

In the summer of 1903 he was able to return to work, and on September 24th, 1903, he reported himself to me. He had been working regularly for several months, and had gained almost complete power in his hands and arms. He felt his legs still weak and the reflexes were still much exaggerated, a slight

tap on the patellar tendon throwing the whole limb into clonic tremor. He was well in every other respect, but felt pain at nape of neck occasionally. There was now no dissociation symptoms, his sense of touch, of pain and of temperature being normal all over.

*Remarks.*—The combination of symptoms in this case pointed undoubtedly to a lesion in the central region of the cord about the lower cervical segment. All above the 7th cervical segment was unimpaired, whilst the skin area, which according to Head is supplied by the 8th cervical segment, was the part affected as regards the sense of temperature and of pain. The dissociation of these senses from that of touch over a definite region of both hands pointed distinctly to the lesion being near or in the central canal of the cord in the position of the lesion in syringomyelia, a disease to which the clinical symptoms in this case corresponded very closely. These symptoms may be briefly summarised as: (1) dissociation symptoms already noted; (2) atrophic paralysis of groups of muscles of hands and arms; (4) spastic condition of lower limbs with increased reflexes.

If the case had been seen for the first time a few weeks after its onset without knowing the history of the case, one would have been justified in concluding that it was a case of syringomyelia as the most typical symptoms of syringomyelia were all present, but the history was too recent for syringomyelia. A healthy young man after exposure and the taking of too much alcohol, suddenly became affected with symptoms pointing to a lesion in the interior of the cord in the lower cervical region. The symptoms pointed to pressure on the fibres in the posterior internal columns at the level of the eighth cervical segment, producing the dissociation symptoms; to pressure on the anterior cornua in the same region producing amyotrophic paralysis of certain muscles of the hands and arms; and to pressure on the antero-lateral columns producing a spastic condition of both legs, with greatly exaggerated reflexes, just as one sees in descending sclerosis. The most feasible explanation of the cause of all these symptoms is, in my opinion, a ~~small~~ hæmorrhage into the interior of the cord, either into or in the neighbourhood of the central canal in the lower cervical region, and the progress of the case is, I think, confirmatory. After a short time the blood extravasated apparently began slowly to be

absorbed, so that the symptoms gradually improved, the nerve cells and nerve fibres recovering their function.

In cases such as this, one can never be sure how much of the symptoms are due to the pressure of the extravasated blood, how much to the laceration of the tissues necessarily produced by its presence, and how much to myelitis set up as a result of its presence. The lesion is, I think, too limited to be due to myelitis alone without hæmorrhage.

Whether the patient will make a complete recovery depends on whether the blood will be completely absorbed, and if so, whether its presence has produced any permanent injury to nerve cells or nerve fibres in its neighbourhood.

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### **PARALYSIS OF THE SERRATUS MAGNUS AND LOWER PART OF THE TRAPEZIUS MUSCLES.**

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IN this paper we give an account of a case of paralysis confined to the serratus magnus and lower third of the trapezius, and make some remarks upon previously reported cases.\*

#### *Description of Case.*

M. C., aged 21, a native of Shetland, a ship-carpenter by occupation, was admitted to Leith Hospital, under the care of Mr Miles, on the 13th of May 1902, suffering from appendicitis. An immediate operation was considered necessary. The abdomen was opened within a few hours of his admission. A gangrenous appendix, perforated at its base, with general peritonitis, was found. The patient made a good recovery from the operation.

On the sixth day after the operation, on lifting his right arm to throw a paper to the patient in the next bed, he found that he was unable to carry out the necessary movement. The right shoulder "felt weak." For two or three days previously he had been using the arms, and up to the morning of the sixth day after the operation he had noticed nothing amiss. Further, on the same day that the weakness attracted his attention he said that he experienced a "numb feeling on the top of the right shoulder," and placed his hand over the right supraspinous fossa to indicate to us the exact spot. This

\* Our best thanks are due to Mr Miles for kind permission to publish this case.

"numb feeling" persisted for two or three days, then disappeared completely. He had never any pain. The patient is an exceptionally intelligent man, and the statements which he made were clear and definite. Previous to this attack of appendicitis, excepting for an attack of pleurisy and pneumonia a year previously and occasional dyspepsia, he said that he had always enjoyed the best of health. The family history was in every respect satisfactory.

The patient was first examined by us on November 8th, 1902, by which time, according to his statement, slight improvement had begun to take place. He was a tall, well developed man, and looked the picture of health. Apart from the condition of the right shoulder, which we are about to describe, a careful examination revealed no evidence of disease. A scar, the result of his recent operation, was present on the anterior abdominal wall in the right iliac region.

As the patient stood at rest, the arms hanging by the side (Fig. 1), there was little to attract the attention of the casual observer. When looked at from the front, nothing abnormal was noticed. On careful observation of the back it was seen that the lower angle of the right scapula was slightly more prominent than its fellow; and that its vertebral border was inclined from above downwards and very slightly inwards, so that it lay almost parallel with the corresponding border of the opposite scapula, its inferior angle lying nearer the middle line than that of the opposite side. The lower border of the greater rhomboid muscle was very distinctly seen on the right side running from the inferior angle of the scapula upwards and inwards towards its vertebral attachment. There was no difference between the level of the tips of the shoulders.

When the arms were extended and held out in front in a horizontal position (Fig. 2), the right scapula projected from the chest in the manner which is so characteristic of serratus palsy. The backward displacement of the scapula became still more prominent when the patient attempted to push forwards against resistance. The vertebral border remained close to and parallel to the spinal column during this movement, and the absence of the serratus digitations in the right as compared with the left axilla was very striking. A band of transverse fibres at the level of the spine of the scapula, i.e. corresponding in position to the middle part of the right trapezius, stood out prominently during this movement. This is not well brought out in the photograph.

When the arms were held out laterally at right angles to the trunk (Fig. 3), the vertebral border of the right scapula, and especially its superior angle, approached the middle line closely, projecting at the same time very markedly from the chest wall. The upper and middle parts of the trapezius and the levator anguli stood out prominently when the right arm was held in this position.

The patient was able to bring his right arm to an angle of about 45° above the horizontal, as may be seen in the photograph (Fig. 4). In making an effort to maintain the arm in this position, the head was inclined slightly towards the left shoulder. The inferior angle of the scapula moved considerably outwards during this movement.

A slight degree of spinal curvature with the convexity to the right is seen in one or two of the photographs. As no note was made as to this point at the time of examination, we shall not refer to it further.

Apart from the defects of movement which were obviously due to the serratus paralysis, no weakness was detected in any of the movements of the right arm or shoulder girdle. Nowhere was there any sign of muscular atrophy.

The faradic excitability of the muscles of the shoulder girdle and upper limb was carefully tested. The right serratus magnus failed completely to respond to a strong faradic current. All the other muscles reacted readily

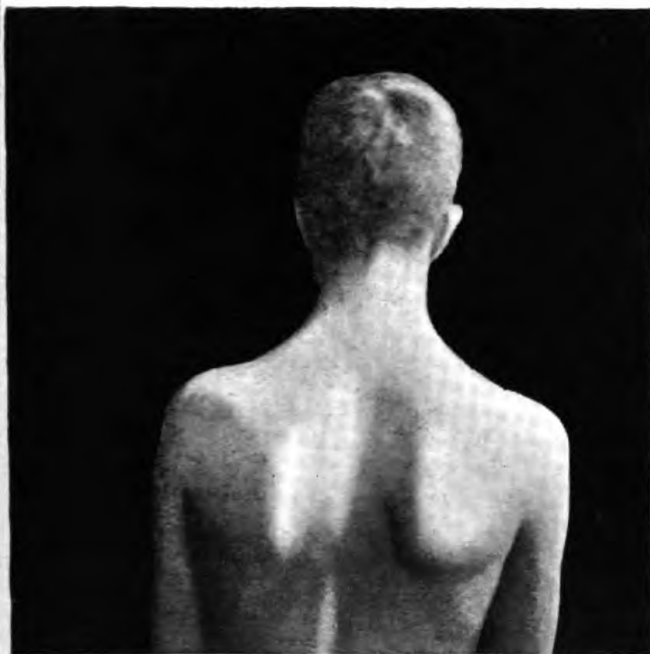


FIG. 1.



FIG. 2.

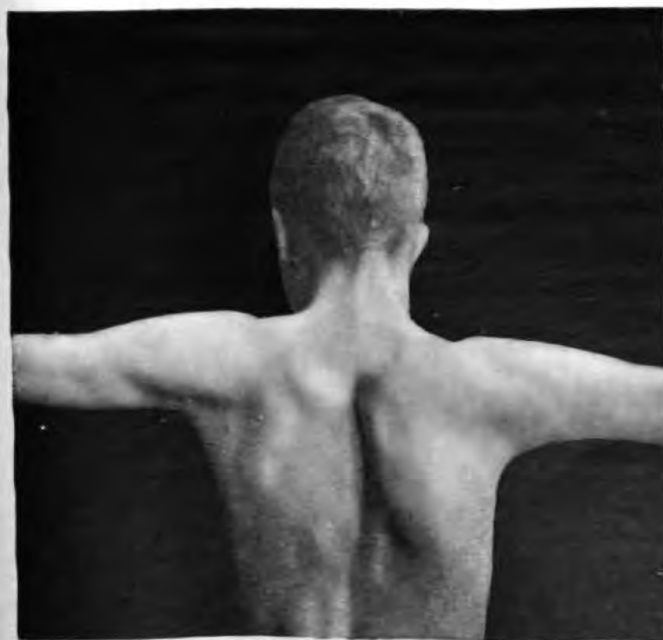


FIG. 3.

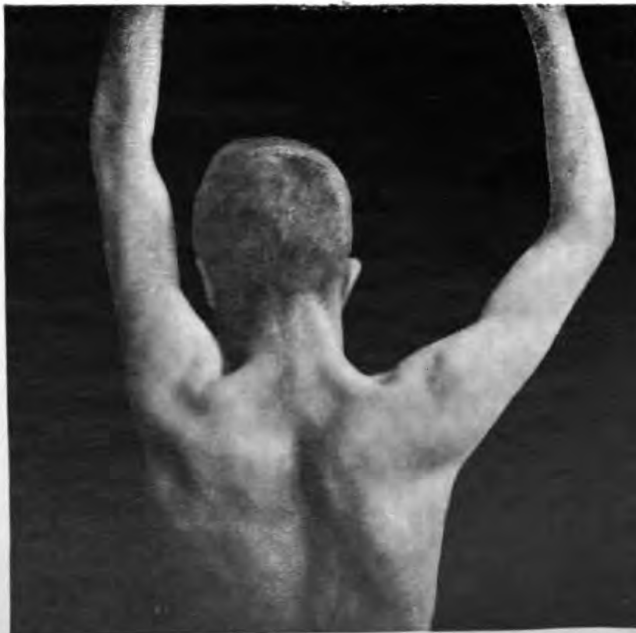


FIG. 4.

Case of Serratus and Trapezius Paralysis described in text.

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2

3

to faradism with the exception of the lower part (ascending fibres) of the right trapezius, in which no response was obtained with a strong current. The whole of the left trapezius and the upper and middle portions of this muscle on the right side responded quite normally to a moderate current. Unfortunately the constant current was not employed. Four months after the onset of the paralysis improvement began to occur.

In February 1903, the only sign of serratus palsy which we were able to detect was slight undue prominence of the right, as compared with the left, scapula, when the patient used his right arm to push forwards against resistance. Even at this date, however, there was slight obliquity of the vertebral border, the lower angle of the right scapula lying nearer the middle line than its fellow.

Eleven and a half months after the patient left hospital he was back at work and felt the right arm in every way as strong as its fellow.

*Summary.*—Let us briefly summarise the chief features of this case. A young and healthy man is seized with an attack of appendicitis, with general peritonitis, for which he undergoes an operation. On the morning of the sixth day after the operation he notices that there is some weakness about the right shoulder, and on the same day he complains of a numb feeling in the region of the right supraspinous fossa. On examination six months after the onset of the paralysis, when, according to the patient's statement, slight improvement had already begun to appear, the following were the most striking positive facts elicited:—(1) With the arms hanging at rest by the sides there was a slight abnormal obliquity of the vertebral border of the right scapula, the inferior angle of the scapula lying nearer the middle line and projecting further from the chest wall than that of the opposite side; (2) when the arms were held out in front at right angles to the body, the "winged scapula" which accompanies paralysis of the serratus magnus was seen to be present in a marked degree; (3) the patient was able to raise the right arm to an angle of  $45^{\circ}$  above the horizontal; (4) there was a complete absence of faradic excitability in the serratus and lower third of the trapezius muscles on the right side. The lesion was probably a neuritis. The absence of pain was unusual. Improvement began to occur four months after the appearance of the paralysis, and seven months later there was no discoverable sign of serratus palsy.

#### *Frequency of Isolated Serratus Paralysis.*

Paralysis of the serratus magnus in association with paralysis of other muscles of the shoulder girdle is not uncommonly seen in cases of progressive muscular atrophy, the myopathies, etc.

Isolated serratus magnus palsy is, on the other hand, rare. Many of the reported examples have been without doubt complicated cases. Thus Duchenne, referring to the numerous instances of serratus paralysis which had been reported prior to 1867, remarks on the frequency with which other muscles were also involved (1). The same writer (2), in 1867, made the statement that although he had seen twenty cases of atrophy or paralysis of the serratus magnus, he had never met with an isolated palsy of that muscle.

Lewinski (3), in a very thorough revisal of the literature previous to 1878, was of opinion that only one case of isolated serratus palsy had been reported up to that time. This was a case described by Busch (*Langenbeck's Archiv. f. klin. Chir.*, 1863, iv. S. 39).

Barreiro, in his Paris thesis ("Contrib. à l'étude de la paralysie du muscle grand dentelé," 1895), which is quoted by Souques and Castaigne (4), was able to collect only sixteen cases from the literature of what he considered to be undoubted examples of isolated serratus palsy.

Steinhausen (5), in 1900, wrote a valuable paper on the subject; after an exhaustive up-to-date study of the literature from the publication of Lewinski's paper in 1878, he selected twenty-nine cases which were, in his opinion, undoubted instances of isolated serratus palsy. In this paper he points out (6) the comparative frequency of cases of serratus paralysis in the German army. During the two decades—1880 to 1900—there were ninety-five instances of serratus paralysis recorded in the army reports, of which Steinhausen extracts sixty as uncomplicated examples. This apparent prevalence among soldiers is of interest, but the figures do not appear remarkable when the great numerical strength of the German army is remembered, together with the much greater frequency of serratus paralysis among males, and especially manual workers. Further, as Steinhausen himself points out, soldiers, unlike the labouring classes, are constantly under medical observation, and therefore in them slight degrees of serratus paralysis are much more likely to attract attention.

*Relative Frequency of Cases of Paralysis confined to the  
Serratus Magnus and Trapezius Muscles.*

Paralysis of the serratus magnus is not uncommonly seen in association with paralysis of other muscles. The muscles most



usually affected are the deltoid, supra- and infra-spinatus, and especially the trapezius (7). A number of cases of paralysis confined to the serratus and trapezius have been recorded. According to Barreiro (8), concomitant trapezius paralysis is the most common complication of serratus palsy. Souques and Duval (9) insist upon the frequency of this association.

We have found references to seventeen cases of paralysis limited to these two muscles. Some of these cases are merely referred to and not described. It is especially the lower and middle trapezius fibres which suffer in this relation ; indeed, with the exception of one instance mentioned by Steinhausen, the upper trapezius seems always to have escaped.

So far as we can ascertain, the first case of paralysis limited to the serratus and trapezius was observed by Duchenne (10). In this case the paralysis involved both the lower and middle fibres of the latter muscle.

Moritz Meyer (11) has described a case of this kind occurring in a tinsmith. In this case both the right serratus and the lower part of the right trapezius reacted more feebly to faradism than the muscles of the left side. The upper and middle portions of the right trapezius and the levator anguli scapulæ on the right side were hypertrophied.

Bernhardt (12) has published a case of combined serratus and trapezius paralysis, no other muscle being affected, but he makes no statement as to the extent of the trapezius palsy.

A similar case has been recorded by Souques and Duval (13), in which the middle and lower two-thirds of the trapezius were paralysed.

Souques (14) has also described another case of the same nature, in which the paralysis had a similar distribution.

Remak (15), in discussing Brun's paper, mentioned incidentally that he had seen six cases of associated serratus and trapezius paralysis and only three cases of isolated palsy of the former muscle.

Among the ninety-five cases of serratus paralysis collected by Steinhausen (16) from the German army reports, there were six cases of paralysis confined to the serratus magnus and trapezius muscles. In four of these cases the lower part, in one the middle third, in the remaining case the upper part of the trapezius was affected.

From these remarks it will be seen that quite a number of cases of paralysis limited to the serratus magnus and trapezius muscles have been observed.

It is quite probable that in some cases of supposed isolated serratus palsy, a paralysis of the lower trapezius may have been overlooked, for, as we shall mention later, paralysis of this part of the muscle appears to cause no additional symptoms, and can only be demonstrated by an electrical examination.

*The Etiology of cases of Paralysis confined to the Serratus Magnus and Trapezius Muscles.*

The serratus magnus receives its nerve supply from the long thoracic nerve, the origin of which from the fifth, sixth and seventh cervical roots is very constant (17). The trapezius is innervated by the spinal accessory and by branches from the third and fourth cervical nerves, which unite with the former under the muscle to form the subtrapezial plexus. The lower and middle thirds of the trapezius are supplied mainly by the cervical branches, the upper portion chiefly or entirely by the spinal accessory. There is no known peripheral anastomosis between the nerve supply of these two muscles. If both the serratus magnus and trapezius are paralysed in consequence of a peripheral nerve lesion, it follows that the nerves of both muscles must be involved.

As has been already stated, only one case is referred to in the literature in which the upper trapezius was paralysed. The escape of the upper trapezius in these combined cases is discussed at considerable length by Souques and Duval (18). These writers bring forward unassailable evidence to show that morphologically, physiologically and pathologically, the upper third of the trapezius is to be regarded as a distinct muscle.

Why are the serratus and trapezius paralysed together? Does a study of the etiological data in the recorded cases furnish any satisfactory explanation to account for the involvement of the two nerves? An answer to these questions necessitates a study of the reported cases. The only available cases for this enquiry are those of Duchenne, Moritz Meyer, Bernhardt, Souques and Duval, and Souques.

Duchenne's patient was a brush-maker, aged 34. There was "atrophy confined to the right serratus magnus, the inferior

third and a great part of the middle third of the right trapezius." The condition was attributed to excessive work ("*par abus de travail*").

The paralysis developed suddenly in Meyer's case after the patient had been working for a long time in a stooping position, and was ushered in by a severe pain in the right supraspinous fossa.

Bernhardt writes of his patient, a female aged 20, that while she was lifting down a heavy object from above her head she felt a severe pain in the right shoulder blade.

Souques and Duval's patient was a boiler-maker, 45 years of age. From their admirable description of the case it appears that the paralysis also came on quite suddenly while the patient was putting forth all his strength to raise and turn over a heavy boiler. "At the moment when he changed his hands for the second part of the movement, and began to push, he experienced a pretty severe pain in the space between the right pectoral and deltoid muscles and in the region of the right supraspinous fossa, and at the same time found that he was unable to raise the right arm to the vertical" (19).

Souques' second patient was a muscular girl 20 years of age. While walking upstairs she missed her footing, and, to save herself from falling, she caught hold of the banister with her right hand, so that all the weight of the body was thrown on the right arm. She felt a pain in the right shoulder, and found that she could not raise the arm above the horizontal.

No positive etiological details are available regarding the twelve cases incidentally referred to by Remak and Steinhausen.

Souques and Duval, in describing their case, point out that in all probability the nerves to the two muscles were injured by the forcible synergetic muscular contraction which occurred at the time the paralysis developed. The probability of this explanation is obvious. The cases described by Bernhardt and Souques, and possibly Meyer's case, admit of a similar explanation. Duchenne's case cannot, however, be so explained.

Steinhausen (20) incidentally mentions that in none of his army cases did the onset of the paralysis follow upon violent muscular effort, but he makes no further reference to their etiology. From this statement it will be seen that all cases are not due to stretching or pressure on the nerves occurring in con-

sequence of a powerful synergetic contraction of the muscles of the shoulder girdle.

Remak (21) has suggested that the proximity of the long thoracic to those cervical branches which are distributed to the trapezius affords a very probable explanation of the combined paralysis.

It will be noticed that in all the five cases which we have referred to the right, serratus and trapezius were affected. Isolated serratus palsy is much more common on the right than on the left side.

So far we have made no reference to our own case in this connection. Injury to the nerves resulting from a forcible synergetic muscular contraction can, we think, be definitely excluded. The weakness in the arm was first noticed six days after an operation for appendicitis, during which time the patient had been lying on his back. Although he had been using his arms for two or three days prior to the time when the weakness was first felt, he noticed nothing amiss until on the morning of the sixth day after the operation, when he found that he was unable to throw with his right arm. We wish particularly to point out that he did not use any force in attempting to throw; he merely lifted up his right arm and found he could not throw. There was no history of direct trauma at the time of the operation or subsequently. It may have been that the paralysis had been present for some days before it was observed. This, however, seems unlikely, for it was on the day that the weakness was first noticed that the "numb feeling" in the region of the right supraspinous fossa appeared.

We know of no case of isolated serratus palsy occurring as a result of a strained position on the operating table.

It is reasonable to suppose that the paralysis in our case was probably dependent on a neuritis in some way connected with the appendicitis and general peritonitis. A number of cases of isolated serratus palsy following upon acute infective disorders have been described. Thus cases have been reported in which the paralysis was a sequel of typhoid (Bäumler and others), influenza (v. Rad), pneumonia (Steinhausen), diphtheria (Seeligmüller), etc. We must assume in our case that for some reason, regarding which we shall not attempt to theorise, both the nerves to the serratus and lower trapezius were predisposed to

suffer from toxæmia associated with the peritonitis which was here present.

*Symptomatology of Isolated Serratus Magnus Paralysis and of Combined Serratus Magnus and Trapezius Palsy in reference to (a) the position of the scapula when the arms are at rest and hanging by the sides, and (b) the ability to raise the arm above the head.*

The scapula muscles may be divided into two great groups, viz. : (1) those which attach the scapula to the trunk, and (2) those which connect the scapula with the upper extremity.

The function of the first group, to which the serratus magnus belongs, is of a twofold nature. These muscles (*a*) move the scapula on the trunk, and (*b*) by their combined action fix the scapula, affording a *point d'appui* from which those muscles which move the arm on the shoulder girdle can act. The serratus magnus is of great importance in both these relations. The special functions of the serratus magnus are to fix the vertebral border of the scapula to the chest wall and to pull the whole scapula, and especially its inferior angle, outwards and forwards.

When the serratus magnus is paralysed, movements such as pushing forwards with the extended arm, throwing, and elevation of the arm above the head, all of which depend upon a forward movement of the scapula, are those which are especially interfered with. Brodman (22) has made the rough calculation that isolated serratus paralysis reduces the average workman's working value by 15 per cent. Obviously, however, the effect upon the working capabilities of anyone affected with isolated serratus palsy will depend upon the special nature of his work.

Does isolated paralysis of the serratus magnus produce any change in the position of the scapula when the arms are hanging at rest by the sides?

With regard to this point Duchenne (23) states : " I have demonstrated that atrophy of the serratus magnus produces no deformity in the position of the shoulder during muscular repose when the arms hang by the sides of the body, except when the lower two-thirds of the trapezius are at the same time entirely atrophic." Berger (" Die Lähmung des Nervus thoracicus longus," Breslau, 1873, S. 43), quoted by Bernhardt (24), writes as follows : " In complete paralysis of the serratus and in complete

integrity of its antagonists, in the position of rest, the arms hanging by the sides, the following deformity shows itself. The scapula slopes with its vertebral border running from above and externally downwards and inwards, its lower angle in particular projecting backwards in a wing-like manner. The scapula lies as a whole higher, and is nearer the vertebral column. The outer border of the scapula takes up a more horizontal position." Lewinski (25), in 1878, mentions that in the only certain case of isolated serratus palsy reported up to that time (the case described by Busch), there was no abnormal position of the scapula when the arms were at rest. Seeligmüller (26), who in 1882 devoted a paper to the consideration of this point, states that in long-standing isolated serratus palsy, in the position of rest, the inferior angle of the scapula always approaches the vertebral column. Remak (27) holds that the oblique direction of the vertebral border from below upwards and outwards is only seen when the lower or middle portions of the trapezius are also involved. Bäumlér (28) and Bruns (29) have reported cases of complete isolated serratus palsy in which a slight degree of the scapula deformity described by Berger was present.

Souques and Castaigne (30), referring to this question, say : "The truth appears to us to lie between the extreme statements of Duchenne and Berger. There is really a displacement of the scapula, but this displacement is moderate and may escape notice on a superficial examination."

Summarising opinions, it appears that when paralysis is limited to the serratus magnus, although slight abnormal obliquity of the posterior border of the scapula, with prominence of the inferior angle, etc., may be present (*e.g.* cases of Bäumlér, Bruns and others), the deformity is always slight.

On the contrary, when the middle part of the trapezius is paralysed in association with the serratus, a marked deformity results, as Duchenne, Berger and others have pointed out. The tip of the shoulder lies at a lower level than normally, the inferior angle of the scapula is displaced inwards and upwards, while the superior angle projects upwards and may even be seen as a distinct prominence above the line of the shoulder when the patient is viewed from the front.

In the four cases referred to where there was a paralysis, limited to the serratus and lower and middle thirds of the

trapezius, this deformity was striking. The abnormal position assumed by the scapula in cases where the serratus and middle trapezius are both affected is probably to some extent due to the weight of the arm, but the unopposed action of the muscles inserted into the coracoid process, of the rhomboids and levator anguli scapulæ may participate in its production.

Where only the lower part of the trapezius is paralysed although, as in our case and that of Moritz Meyer, there may be a slight abnormal obliquity of the vertebral border and slight prominence of the inferior angle, the alteration in the position of the scapula does not appear to be more marked than in some reported cases of isolated serratus palsy. The carefully recorded cases of Bäumlér and Bruns show that the slight obliquity of the vertebral border does not depend on weakness of the lower trapezius, for this muscle was not involved in their cases. The prominence of the rhomboids in our case (Fig. 1) suggests that the unopposed tone of these muscles may perhaps aid in producing the obliquity.

Inability to raise the arm above the horizontal has been insisted upon by many writers as an important sign of serratus paralysis. Thus Souques and Castaigne say: "Dans la grande majorité des faits, l'élévation du bras n'atteint pas ou atteint exactement la ligne horizontale, mais ne la dépasse pas" (31).

Steinhausen, however, who paid especial attention to this point in his revision of the literature, concludes that "Die Erhebung des Armes über 90° hinaus ist bei der isolirten Serratus-Lähmung nicht die Ausnahme sondern die Regel" (32). In 18 of the 29 cases of isolated serratus palsy collected by him from the general literature (33), and in 57 of the 60 army cases elevation above the horizontal was possible.

A few remarks regarding elevation of the arm and the muscles which are associated in the movement are necessary. Duchenne has shown that electrical stimulation of the deltoid produces an elevation of the arm almost to the horizontal (34). The arm cannot be raised above this level unless the scapula is rotated. The important muscle in connection with this rotation of the scapula is the serratus. Duchenne found (35) when he stimulated the serratus and the deltoid at the same time that the arm was raised to the vertical with great force. The movements of abduction of the arm and rotation of the scapula occur more or

less simultaneously, as anyone may demonstrate for himself. Duchenne has also shown (36) that the middle part of the trapezius together with the deltoid may produce an elevation of the arm to the vertical, though with much less force than the serratus. Later (page 70), he adds that when the serratus is paralysed the middle part of the trapezius must be well developed and act with force in order to produce elevation of the arm above the horizontal. The inability to raise the arm above the horizontal in the recorded cases of paralysis of the serratus and middle part of the trapezius is of interest in this connection, as is the hypertrophy of the middle and upper parts of the trapezius and of the levator anguli in Meyer's case. In our case, as we have stated, the middle and upper trapezius stood out prominently when the patient attempted to raise his arm to the vertical.

From the above considerations it would appear probable that where the serratus is completely paralysed, elevation of the arm to the vertical is only to be expected where the middle part of the trapezius is particularly well developed. The different degrees of muscularity met with in different individuals may account for varying degrees of elevation which are possible in different cases.

Further, if the paralysis does not affect the whole serratus, or if it is only slight in degree (a paresis), differences in the power of elevation of the arm will no doubt occur.

Steinhausen (37) has advanced an ingenious explanation to account for the ability or inability to raise the arm to the vertical in cases of serratus palsy. This writer holds that in all cases in which elevation to the vertical is possible, the upper part of the serratus has escaped. As he points out, it is impossible to examine the upper fibres of the serratus on account of their position. Steinhausen quotes Jolly's case in support of his theory. The patient was a young girl who received a stab in the axilla, and, in consequence, a paralysis of the serratus magnus. The patient, who was not a muscular subject, could raise her arm to the vertical. Steinhausen argues, from the position of the stab, that the branches to the upper part of the muscle had escaped injury, and he concludes that elevation of the arm was produced by the upper part of the serratus, which presumably escaped. Steinhausen does not hold that the upper serratus fibres produce the rotation of the scapula, but that they afford a fixed point on



which the middle fibres of the trapezius can act. One must conclude from this case that the upper part of the serratus is of importance in producing elevation of the arm to the vertical.

Further, Steinhausen refers to another case recorded in the German army reports. The patient, a soldier, received a stab above the clavicle, which produced as a result paralysis of the serratus. He was only able to raise his arm slightly above the horizontal. Steinhausen believes that in this case the branches to the upper part of the muscle were also paralysed.

Steinhausen lays special emphasis on the liability of the fibres to the upper serratus to escape injury on account of their anatomical relations. He states that the fibres to the upper part of the serratus are usually given off from the nerve high up soon after it has pierced the scalenus medius muscle. Struthers' observations corroborate this statement, but show that they run down on the scalenus medius parallel to and in close relation to the trunk formed by the union of its upper two roots as far as the first rib, so that it is very difficult to conceive of any injury to the nerve above the clavicle at least which would injure this trunk and leave untouched the branches to the upper digitations of the muscle. It seems to us that the lower root of the posterior thoracic nerve, which arises from C. 7, and which supplies the lower part of the muscle, is much better protected in the neck than the roots from C. 5 and C. 6.

Steinhausen states that the branches to the upper part of the serratus lie underneath the muscle, and enter it from its internal surface, and hence are especially well protected from injury. Dissections made by one of us (J. W. S.) show, however, that these branches to the upper digitations enter from the outer and not from the inner aspect of the muscle.

It must be granted that the upper fibres of the serratus are of importance in elevating the arm to the vertical (Jolly's case), and that in some of the reported cases of serratus palsy the upper part of this muscle may have escaped; the evidence, though most suggestive, does not, however, appear to us to be sufficiently conclusive to justify the absolute statement that in all cases of serratus palsy where the arm can be raised to the vertical, the upper serratus fibres have escaped.

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31. *Loc. cit.*, p. 193.
32. *Deutsche Zeitschrift f. Nervenheilk.*, Bd. xvi., 1900, S. 425.
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35. *Loc. cit.*, p. 58.
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**THE ANATOMY OF THE LONG THORACIC NERVE,  
WITH SPECIAL REFERENCE TO PARALYSIS OF  
THE SERRATUS MAGNUS.**

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IN discussing the etiology of isolated paralysis of the serratus magnus, many authors lay stress on the alleged fact that the position and course of the long thoracic nerve expose it specially to injury. Its position in the neck is said to render it liable to be injured by direct pressure, blows, or wounds in the supra-clavicular region, and its relation to the scalenus medius muscle is said to render it liable to be injured by violent or prolonged action of that muscle.

Thus Gowers and Taylor (1) say: "in consequence of its position and long course, the nerve to the serratus often suffers." "It may be damaged in the neck by direct pressure—as in carrying on the shoulder a heavy sharp cornered object—or by severe muscular effort, *e.g.* long continued effort with the raised arm." "In such cases a traumatic neuritis is set up by the violent compression of the nerve consequent on the forcible or prolonged contraction of the muscle through which it passes." Oppenheim (2) says: "the nerve may be damaged by trauma in the supra-clavicular fossa." "The carrying of a heavy load may act as trauma." "Further, it is possible that the nerve may be compressed by violent contraction of the scaleni."

Bernhardt (3), in discussing serratus palsy, expresses similar views. Merkel ("Topograph. Anat.") (4), after describing the anatomy of the long thoracic nerve, states that its position after it emerges from the scalenus medius is very superficial, and that in violent movements it may be easily torn or subjected to severe pressure, in its course through the muscle.

Although the anatomical relations of the nerve are in this way held to explain its liability to injury, these relations are in the works referred to, incorrectly described. Thus Gowers and Taylor (1) say: "After being formed in the substance of the scalenus medius muscle by the union of branches of the 5th and 6th cervical nerves, it (the long thoracic nerve) passes behind the brachial plexus and along the side of the chest to

the lower border of its muscle." Oppenheim (2) states that the nerve commonly arises by two roots, from the 5th and 6th cervical nerves, and occasionally by a root from the 4th nerve as well.

Merkel states that the nerve arises by three roots from the 5th, 6th and 7th nerves, and that the three roots pierce the scalenus medius muscle. He states further that the 5th root supplies the two upper digitations of the muscle, the 6th root the next two, and the 7th root the remainder. Bernhardt does not describe the anatomy of the nerve, but seems to imply by his remarks on the etiology of serratus palsy that the entire nerve pierces the scalenus medius.

In Quain's Anatomy (5), on the other hand, the nerve is described as arising by three roots, from the 5th, 6th and 7th cervical nerves, the upper two roots piercing the scalene muscle and the lowest root passing in front of it. As varieties, are mentioned the occasional absence of the root from the 7th nerve, the occasional presence of a root from the 8th nerve, and the occasional distribution of the 5th root to the upper part of the serratus, without any junction between it and the rest of the nerve. In Morris's Anatomy (6), and Cunningham's Practical Anatomy (7), the nerve is similarly described.

There is thus some discrepancy between the various descriptions of the nerve: first, as regards the normal number of roots; and, second, as regards the relation of the roots to the scalenus medius muscle.

The writer has made twenty-five dissections of the long thoracic nerve with a view of ascertaining: (1st) the usual origin of the nerve; (2nd) the usual relation of the various roots to the scalenus medius muscle; and (3rd), the bearing of the ascertained facts on the possibility of isolated injury to the nerve. Nineteen dissections were made in adults, one in a subject of seventeen years of age, two in subjects below ten years of age, and three in fetuses at or near full time. The dissections were made in recent subjects in which the colours of the parts were well preserved and the presence of fine nerve twigs easily detected. The method followed was first to expose the nerve in the supra-clavicular region by reflecting the integuments and removing the fat in the lower part of the posterior triangle of the neck. After noting the relation of the nerve to the main

trunks of the brachial plexus and the clavicle, the muscles attached to the clavicle were divided and the bone thrown back at the acromio-clavicular joint. The two pectoral muscles and the omo-hyoid were then reflected, and the further course of the nerve in the axilla identified. The cords of the plexus were then divided well away from the chest wall and turned aside, in order to get a complete view of the posterior thoracic nerve in its whole course and to ascertain the points of origin of its roots from the main cervical nerves. The main nerves were then divided above the origin of the roots of the long thoracic nerve and removed along with the nerve for reference.

In seventeen of the subjects examined the nerve was found to have a normal origin and course, which may be described as follows :—The nerve arises by three roots, from the fifth, sixth and seventh cervical nerves. The upper root arises from the posterior aspect of the fifth nerve in common with the nerve to the rhomboids, just as it leaves the groove in the transverse process of the fifth cervical vertebra, and passes immediately into the substance of the scalenus medius muscle, running downwards, with a slight inclination backwards, towards the surface of the muscle. In its course through the muscle or just after it appears on the surface, it joins the root from the sixth cervical nerve.

The middle root has a precisely similar origin and course, from the sixth cervical nerve.

The trunk formed by these two roots appears on the surface of the scalenus medius from 3·5-5 cm. above the clavicle, and from a few mm. to 2 cm. behind the line of emergence of the cords of the brachial plexus from between the scalene muscles. It runs down on the scalenus medius, and while in this position gives off several fine branches to the upper part of the serratus magnus muscle, which enter the outer aspect of the muscle.

The origin of these branches is distributed over this portion of the nerve as it lies on the scalene muscle, *i.e.* they do not all arise together, but at different levels. Thus when the two roots do not join till after they pierce the scalenus medius, one of these branches may leave the fifth root just before it joins the sixth root. The remainder of the branches, generally two or three in number, come from the portion of the nerve formed by the union of these two roots as it runs down on the muscle. Occasionally

a branch to the upper serratus arises after the lowest root, from C. 7, joins the remainder of the nerve on the serratus.

As the trunk formed by the two upper roots reaches the insertion of the scalenus medius it passes from view behind the cords of the brachial plexus. When the two upper cords of the plexus are pulled well forward, the trunk is seen to pass from the scalene muscle on to the serratus magnus, and the third and lowest root from the seventh cervical nerve is brought into view. This root arises from the back of the seventh nerve as it passes in front of the scalenus medius, and like the main trunk, runs in front of the scalene muscle. It joins the remainder of the nerve at or slightly below, the upper border of the serratus. In its separate course it is under cover of the upper cords of the plexus, and its junction with the remainder of the nerve takes place under cover of the plexus. It gives off no branches before joining the rest of the nerve. The completed nerve runs down on the serratus magnus to its lower border, lying about two inches behind the line of emergence of the lateral branches of the intercostal nerves, and giving off branches to the muscle as it goes.

These facts may be summarised as follows:—The nerve arises by three roots. The two upper roots pass through the scalenus medius and unite with each other in the muscle or on its surface. The trunk of union supplies the upper and part of the lower portion of the serratus. This trunk and its branches to the upper serratus are exposed on the scalenus medius for a short distance in the neck. The lowest root does not pass through the scalene muscle, and is not exposed in the neck. It supplies in part the lower portion of the serratus.

In eight of the cases examined variations from the normal were found. These were as follows:—In one case there was no root from the 7th nerve. The other two roots had a normal origin and course. In the remaining seven cases the variation from the normal was found in the relation of the two upper roots to the scalenus medius. Thus in one case the two upper roots both passed in front of the muscle. In a second case the upper root came off in two portions, arising close together: one of these passed through the scalenus medius, while the other passed in front of the muscle. In this case the middle root from the 6th nerve also passed in front of the muscle.

In two cases the middle root from the 6th nerve passed in front of the scalenus medius.

In one case the 6th root came off in two portions: one of these pierced the muscle while the other passed in front of it.

In one case the root from the 5th nerve passed through the muscle, but did not join the rest of the nerve, and was distributed entirely to the upper part of the serratus. The 6th root in this case passed in front of the muscle and gave branches to the upper part of the muscle as well as the 5th root.

In one case a small muscular slip which passed from the middle to the anterior scalene crossed in front of the root from the 6th nerve, which passed in front of the rest of the muscle.

Of the varieties mentioned by Quain (1), the only one which was not found was the presence of a root from the 8th cervical nerve.

The writer has not found any reference in the literature to the fact that the 6th root frequently, and the 5th root occasionally, pass in front of the scalenus medius.

From a consideration of the above facts it appears :—

A. When the nerve is normally arranged :—

1st. The whole long thoracic nerve is never collected into one trunk which could be picked out by, say, a small punctured wound in the neck; for the branches to the upper part come off before the lowest root joins the rest of the nerve, and the lowest root lies in the neck under cover of the upper cords of the brachial plexus.

2nd. The portion of the nerve which is exposed to injury in the neck, and which might conceivably be damaged by violent action of the scalenus medius, is the trunk formed by the union of the two upper roots of the nerve. This trunk lies on the scalene muscle covered only by the integuments, by the fat in the supra-clavicular fossa, and by the fascia on the muscle. While in this position it gives off the twigs supplying the two upper digitations of the serratus. These twigs run close to their trunk of origin on the scalenus, and it is difficult to see why, as Steinhausen (8) suggests, they should escape injury when the rest of the nerve is supposed to be damaged.

3rd. The lowest root of the nerve, which supplies in part the lower digitations of the muscle, lies under cover of the brachial plexus in the neck and does not pierce the scalenus medius. It

therefore will escape bruising in overaction of that muscle, and is the portion of the nerve most likely to escape direct injury in the neck.

4th. In connection with the theory that the long thoracic nerve may be damaged by violent or prolonged action of the scalenus medius, it should be remembered that the nerve to the rhomboids comes off along with the upper root of the long thoracic nerve, and, like it, pierces the scalene muscle. If the theory were correct, one would expect that, in some cases at anyrate, the rhomboids would be paralysed along with the serratus. There is no case recorded, so far as the writer is aware, of paralysis limited to the serratus and rhomboids.

*B.* When the nerve is abnormally arranged :—

1st. In the rare cases in which the seventh root is absent, the whole nerve is more liable to isolated injury in the neck than when the nerve is normally arranged.

2nd. In the cases where one, or both, of the upper roots runs in front of the scalenus medius, these roots lie in their course in the neck under cover of the main nerves from which they arise ; and in such cases, therefore, the nerve is less likely to be picked out by injury in the neck than in cases in which it is normally arranged.

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## Abstracts

### ANATOMY.

#### **REPORT ON THE PROGRESS OF ANATOMY OF THE CENTRAL (359) NERVOUS SYSTEM DURING THE YEARS 1901 AND 1902.**

L. EDINGER and A. WALLENBERG, *Schmidt's Jahrb. der ges. Med.*,  
Bd. cclxxix.

IN these pages Edinger with his collaborator has reviewed the publications on the anatomy of the nervous system which have appeared within the past two years, in the same objective yet critical manner as he has already treated the work of the preceding fifteen years.

It is a matter for regret that such capable and reliable work is so little used by those interested in, or at work on, this subject, for the successive reports give, in as succinct a form as is compatible with accuracy and necessary detail, the results of all previous workers in this domain, and a perusal of their pages would check the superfluous accumulation of literature and of "new discoveries" of facts already known and often needless of corroboration.

The present report deals with 632 papers which appeared during the years 1901 and 1902. And as the paucity of new or important results from this mass of work is certainly justification for strong condemnation of the indiscriminate publications with which we are to-day inundated, it makes the more valuable the collection of concise and critical abstracts now under notice. Its most admirable feature is the objective manner in which the work has been done and the careful emphasis laid on any new facts.

A section is devoted to each branch or subdivision of the subject, and an enumeration of these best serves to indicate the nature of the work:—Text-books; Methods; Ganglion Cells, the latter section including notices of 130 papers; The Fore-brain; Cerebral Cortex; "Tween-brain and Mid-brain; Long Tracts; Cerebellum; Pons Varolii and Medulla; Cord; Spinal Ganglia, etc.

And there is besides a full review of the recent work on the comparative anatomy of the brain, a branch of neurology which Edinger rightly insists is not used enough in our constant striving to obtain such an anatomical knowledge as will enable us to more fully understand the correlation and method of working of the nervous system.

GORDON HOLMES.

**A CONTRIBUTION TO THE STUDY OF THE COURSE OF SOME  
(360) SPINAL TRACTS.** L. V. DYDYNski, *Neurolog. Centralbl.*,  
October 1, 1903, p. 898.

THE details furnished in this paper were obtained on examination by Marchi's method of the central nervous system of a case of myelitis in the fifth and sixth dorsal segments, of five months' duration.

Results that—

- (1) Some of the fibres of the dorsal columns pass direct through the corpora restiformia to the cerebellum.
- (2) A few fibres of the dorsal part of the direct cerebellar tract end in the nuclei of the dorsal columns. There are intramedullary portions of the dorsal roots which lie in the lateral and not in the dorsal columns, but are of the same nature as the fibres of the latter.
- (3) Some fibres of Gower's bundles seem to end in the inferior olives. Others join the corpora restiformia.
- (4) Gower's bundle ends—
  - (a) By sending fibres to the velum medullare anticum, which decussate in it and probably pass to the cerebellum.
  - (b) In the corpora quadrigemina.
  - (c) Some fibres join the median fillet and run to the thalamus.
  - (d) A few fibres also seem to pass through the middle cerebellar peduncle to the cerebellum.
- (5) Degenerated fibres in the ventral columns of the cord among the outgoing ventral roots pass upwards to end in the inferior olives. These probably represent Helweg's tract (Olivenbündel, Bechterew).
- (6) Immediately below the lesion the comma tract was found degenerated, and also the "oval field" in the lumbar region, and the "triangular field" (of Gombault and Phillipe) in the sacral cord. The latter two are probably only different portions of an endogenous system and independent of the comma tract.

GORDON HOLMES.

**THE AURIC REACTION AND INTERNAL STRUCTURE OF THE  
(361) NERVE CELLS OF THE HUMAN SPINAL GANGLIA.**  
ENRICO ROSSI, *Névraxe*, Vol. v. F. 2, p. 189.

By his gold chloride method, the author has demonstrated in human spinal ganglion cells appearances similar to those above described.

The nerve cells are invested by a slightly granular substance

forming in section a ring around the cell. Immediately inside this there is a reticulum composed of fine fibres forming meshes which are larger towards the cell periphery. In the spaces, here and there, are black granules. In many cells where much shrinkage has taken place there are long and short bands having a structure apparently similar to that of the cell investment. These bands pass outwards from this granular zone into the cell capsule.

The reticulum merges into the peripheral granular zone, but never passes outwards beyond it. In sections the network at times is seen passing over the nucleus, thus obscuring its detail of structure; at other times it is arranged around it.

DAVID ORR.

**THE AURIC REACTION AND THE INTERNAL STRUCTURE OF  
(362) THE NERVE CELLS OF THE HUMAN SPINAL CORD.**

ENRICO ROSSI, *Nervaze*, Vol. v. F. 2, p. 173.

USING a special gold chloride method of his own, the author has demonstrated the following structures in the nerve cells of the human cord. In both small and large cells there is an intracellular fibrillar network, composed of fine fibres forming meshes of various shapes and which are narrower around the nucleus.

Scattered irregularly in the meshes of the network and also at the points of junction of the fibres are small deeply coloured particles.

There is apparently a second inner reticulum of fine fibres, but Rossi is unable to state whether any connections exist between this and the one above described. In no instance is the reticulum continued to the surface of the cell or into the processes, but from the meshes fibrils pass into the axis cylinder, remain undivided and run parallel to its long axis. With regard to its relationship with the nucleus, one finds that sometimes the network passes over the nucleus, while at other times the meshes are densely packed around it. The reticulum has no connection with the surrounding tissues and is bounded peripherally by a very dark granular layer which extends around the whole cell. Rossi regards this granular substance as an investing membrane, and from it—in many cells—he has observed dark, granular, fringe-like processes passing out to lose themselves in the surrounding tissues.

DAVID ORR.

**A NEW METHOD OF STAINING MEDULLARY SHEATHS.**

(363) EUGEN FRAENKEL, *Neurolog. Centralbl.*, Aug. 16, 1903, p. 766.

THE author claims for his method certain advantages, especially in the examination of the cerebral cortex, in which not only the tangential but the underlying supra-radial fibres can be easily distinguished. In addition to staining the medullary sheaths a deep

blue-black nuclear elements also present a light blue colour, so that orientation is facilitated, and the necessity for a special nuclear method is done away with. Moreover, sections prepared by this process can, if desired, be also treated by Gieson's fluid for further contrast.

The parts of the nervous system for examination are fixed and hardened in Muller's fluid or in Weigert's potassium bichromate—chrome-alum solution. Pieces are embedded in celloidin, and sections from these are placed for twenty-four hours in Unna's polychrome methylene blue, which can be obtained ready-made from Grüber. The sections are then washed in distilled water and differentiated in an old saturated watery solution of tannic acid. Almost complete decolorisation is allowed to take place, and then, after washing again in distilled water, the whole process of staining and differentiation is gone through again in the same manner, the decolorisation being carried only to the requisite degree. The sections are then passed through 96 per cent. spirit, cleared in Bergamot oil and xylol, and mounted in balsam. The same methylene blue solution (after filtering) is used for the second as for the first staining, and the author of the method believes that the tannic acid in the tissues increases their affinity for the dye.

E. FARQUHAR BUZZARD.

## PHYSIOLOGY.

**THE AUTONOMIC NERVOUS SYSTEM.** J. N. LANGLEY, *Brain*, (364) Spring 1903.

THE autonomic nervous system is the name given by the author to the general group of nerves which controls the unstriated muscle, the cardiac muscle, and the glandular tissue of mammals. The efferent autonomic nerves leave the central nervous system in four distinct regions:—

1. A region underlying the anterior corpora quadrigemina from which fibres pass by the ciliary nerves to the iris and ciliary muscles. This may be called the *mid-brain autonomic system*.
2. A region a little above and below the calamus scriptorius from which fibres pass by the nerve of Wrisberg, the glosso-pharyngeal, and the vagus to supply the mouth end of the gut and structures developmentally connected with it. This may be called the *bulbar autonomic system*.
3. A region in the spinal cord from the first thoracic segment to the second or third lumbar segment inclusive in man. This gives origin to the efferent fibres of the *sympathetic system*.



4. A region between the first and third sacral segments which furnishes the nerve supply of the anal end of the gut and structures developmentally connected with it. This is the *sacral autonomic system*.

Of these four origins the bulbar and sacral most closely resemble one another. The cranial and sacral autonomic systems are not simply separated parts of the sympathetic system, but are probably independent nervous developments with a different history though they may have developed from homologous parts of the central grey matter. The sympathetic system sends fibres to the areas supplied by the bulbar and sacral autonomic systems as well as to parts not supplied by these, and wherever this double nerve supply exists the nerves from either system do not necessarily produce different effects; but if the effect is different, then all the fibres of the one agree in producing an effect different from that produced by all the fibres of the other.

The four systems, however, have many points of resemblance to one another.

1. No efferent autonomic fibre runs from the central nervous system to muscle or gland without having a nerve cell in its course; they are all pre-ganglionic fibres on leaving the central nervous system, and end in connection with nerve cells in ganglia. The ganglia then give off post-ganglionic fibres which end in the tissues. This has been proved by the nicotine method. The ganglia contain cells which give off all kinds of nerve fibres; the cells are not grouped into collections for some special function, but for the supply of definite areas. In the cranial and sacral autonomic systems the ganglia are usually near the tissue they supply; in the sympathetic system they are near some of the tissues they control but far from others. The skin supply of the sympathetic fibres corresponds in the main with that of the spinal nerve which they join; they slightly overlap in the skin of the body wall, but widely overlap one another in the limbs. There are certain parts of the body in which the sympathetic system has little control, and in some, for example, the region of distribution of the mid-brain autonomic nerves, it is doubtful whether the sympathetic has any effect.

2. Most probably all nerve fibres which run from the central nervous system to a ganglion divide into two or more nerve branches.

3. Each branch of a pre-ganglionic nerve fibre is connected with a nerve cell.

4. The post-ganglionic nerve fibre does not in any case run to another nerve cell of the system, but runs to the peripheral tissue, branching as it goes to supply a number of peripheral tissue cells.

5. All the nerve cells of the ganglia are on the course of efferent

fibres; the peripheral ganglia of these systems have no sensory cells, the nerve cells of the afferent fibres being situated in the spinal ganglia.

The "reflex" actions described as occurring in peripheral ganglia may be called *pre-ganglionic axon reflexes*, and are due to impulses set up in one branch of the pre-ganglionic fibre passing to the other branches, and so to tissues more or less remote from the point stimulated; in no case are they set up by an afferent neuron acting on an efferent neuron; they are not true reflexes. *Post-ganglionic axon reflexes* are examples of another form in which there is a spreading out of an impulse from one branch of a peripheral neuron to its other branches. There is no evidence to show that an impulse set up in an autonomic motor nerve cell can pass back through the cell and so stimulate adjoining cells in the ganglion.

In all probability the autonomic nervous system agrees in all essential points in other vertebrates with that existing in mammals. Since all the autonomic nerve fibres proceeding from the central nervous system have nerve cells on their course, it follows that no autonomic nerve fibres run outwards in the somatic branches of the nerves, as no nerve cells occur on these. No autonomic effect was obtained by stimulating a number of nerve roots in the cat. These conclusions are in opposition to results obtained by Stricker and others, who alleged the presence of vaso-dilator fibres in the posterior roots of the nerves which supply somatic fibres to the hind limb of the dog. The author believes that these vaso-dilator effects may be obtained by the spreading of the stimulus to the muscle spindles, and this only holds under certain conditions.

The bulbar autonomic, the sacral autonomic, and the sympathetic nerves just peripheral to the posterior root ganglia, all contain afferent fibres. The trophic centres of these afferent fibres are probably all to be found in the posterior root ganglia. The afferent autonomic and the ordinary afferent nerves present some differences in their physiological action. The viscera are comparatively insensitive under normal conditions; this may be due to the "threshold" for sensation in the autonomic fibres being higher, or more likely to fewer afferent fibres running to a given area. It is difficult, however, to separate the afferent autonomic nerves from the ordinary afferent nerves. They may for the present be arranged into autonomic and somatic divisions by considering as afferent autonomic fibres those which give rise to reflexes in autonomic tissues, and which are incapable of directly giving rise to sensation, and by classing all other afferent fibres as somatic. On this hypothesis there must be a difference in their central connections, the autonomic path to the cerebral hemispheres must be absent or only very slightly developed.

PERCY T. HERRING.

**THE NATURE OF INHIBITORY PROCESSES WITHIN THE  
(365) NERVOUS SYSTEM.** W. M'DOUGALL, *Brain*, 1903, p. 153.

THE first part of this paper is an examination of various views, more or less current, as to the nature of inhibitory processes in the nervous system. The conclusion is reached that only two views are at all tenable: that inhibition may consist in either (1) a direct checking of the katabolism of the nervous substance, or (2) in the cutting off of nervous influences that cause increased katabolism. It is then pointed out that of the two great divisions of the nervous system, the visceral system, on the one hand, has to control organs that exhibit great capacity of spontaneous activity, and accordingly send to those organs, nerves of purely inhibitory function; on the other hand, the vastly preponderating part of the nervous system is concerned in the control of the skeletal muscles, in which the capacity of spontaneous activity is minimal, and in which, accordingly, inhibition seems to be effected solely by the withdrawal of excitatory impulses. This having been shown to be the case in the lowest sensory-motor arcs of the "voluntary" system, it becomes probable that the process of inhibition is of similar nature throughout the later outgrowths of those most primitive and fundamental parts of that system, *i.e.* throughout the great mass of the higher brain. If we adopt the alternative view, that inhibition consists in a direct checking of katabolism by special "inhibitory impulses," we must assume either (1) very extensive systems of neurones of purely inhibitory function, hardly, if at all, inferior in extent and complexity to the systems of neurones of excitatory function (and of such systems we have no evidence); or (2) that almost every neurone and nerve is capable of carrying two kinds of impulse of opposite type, excitatory and inhibitory. It is then shown that there are very serious objections to the latter assumption; that in no case has it been proved that the impulse transmitted by an inhibitory nerve, such as a vaso-dilator nerve, differs essentially from the ordinary impulse of excitatory nerves; that the available evidence strongly supports the view that the impulses of nerves of the two opposite functions are of the same character, the difference in their effects depending upon a difference in the character of their connections with the organs in which they terminate; that the great variability of the relations of inhibition and augmentation exhibited by the various intimately connected tracts (an excitation-process in one tract spreading at one time as excitation to a neighbouring tract and at another time as inhibition to the same tract) seems to be inexplicable if we assume inhibition to be effected by impulses of a special nature opposite in character to the excitatory impulses.

These considerations seem to show, by a process of exclusion,

that inhibition throughout all that vastly preponderating part of the nervous system which controls the skeletal muscles must consist simply in the withdrawal of excitatory impulses. When we attempt to conceive the mechanism by which such withdrawal is effected, we find clear guidance in the facts of movement of the attention. The giving of the attention to one object results always in the withdrawal of it from the object attended to in the previous moment, and this fact suggests that the coming into activity of one organised system of upper-level brain-paths means that they become at that moment the channel of least resistance for the escape to the efferent side of the nervous system of the energy constantly liberated in the afferent side, under the influence of the ever-varying constellation of sensory stimuli; and that any one organised system of paths in thus becoming the channel of least resistance diverts to itself the main stream of energy, draining it away from the system of paths in which it flowed in the previous moment. This is the hypothesis of inhibition by drainage. It is then pointed out that this seems to be the only hypothesis which will account for the universality of the relation of reciprocal inhibition obtaining between the organised systems of paths of the upper brain-levels; and that the relations of reciprocal inhibition obtaining between different paths of the sensory-reflex, and of the purely reflex, level, are very similar to those of the higher levels and must be regarded as essentially of the same nature. There is then suggested a simple scheme of the mechanism of inhibition by drainage, in terms of which we may readily describe how and why the excitation of one tract commonly leads to inhibition of others in all these levels. And it is shown that the part played by fatigue in determining the hitherto puzzling variation in the relations of reciprocal inhibition is rendered intelligible by this scheme. The paper concludes with the description of some experiments in visual sensation which seem to show that the excitatory processes of the inhibiting tract gain in intensity at the expense of the processes of the inhibited tracts, which must be the case if the hypothesis of inhibition by drainage is true.

AUTHOR'S ABSTRACT.

**QUALITATIVE DIFFERENCE OF SPINAL REFLEX CORRESPONDING WITH QUALITATIVE DIFFERENCE OF CUTANEOUS STIMULUS.** C. S. SHERRINGTON, *Journ. of Physiol.*, Vol. xxx. p. 39.

IN a dog in which a segment from the posterior cervical region of the spinal cord had been previously removed, the author found that when the skin underneath and between the toe-pads and



cushion of the hind-foot was pressed firmly or stretched, and often also at the moment the pressure or tension was removed, a sudden forcible extension of the limb was evoked. If, on the other hand, the skin of the same region was pricked, or if heat sufficient to threaten injury to the skin was applied to it, the limb was immediately flexed or withdrawn from the harmful stimulus. Thus, two stimuli of different quality applied to the same region called forth respectively two reflex movements of exactly opposite sense. "Two different sets of afferent nerves belonging to this part must, therefore, be directly connected with respectively two opposed elements of the muscular organisation of the part."

Another reflex movement capable of being elicited in this "spinal" dog was the "scratch"-reflex. This was obtained when the finger-tip or a pencil-point was drawn lightly along the surface, but not when firmer pressure was made, so that the skin was moved to and fro over the subcutaneous tissue; that is to say, it could be obtained by superficial but not by deep stimulation. It was further proved that the nerve-endings which evoke this reflex lie quite superficial, by the fact that ablation of the surface of the skin to the depth of 6 mm. (not including the deepest ends of the hair-follicles) was sufficient to abolish the reflex from that area. It was also shown that the set of end-organs provoking the "scratch"-reflex in response to purely tactual stimuli was distributed in close relation to hairs.

While stimulation of the deep skin nerve-endings was found to be incompetent to evoke the "scratch"-reflex, the question arose as to whether among the superficial cutaneous nerve-endings those peculiar to the hairs were the only ones efficient. Various "warm" and "cold" stimuli were tried and found ineffective, but a *scratching*-point dragged along the surface excited it readily. This might be due to the fact that in the latter case *nocipient* nerve-endings were stimulated—a term applied by the author to those endings which respond only when harm is threatened to the skin. In proof of this, he found that when mechanical or thermal stimulus was applied to the skin with sufficient intensity to make it actually noxious or harmful the "scratch"-reflex was readily elicited, while it was not so when the stimulus was less intense. He argues, therefore, that among the superficial nerve-endings in the skin, the *nocipient* as well as those merely of the hairs, are competent to elicit the "scratch"-reflex, but that the "cold" and "warmth" end-organs are not competent.

The following general conclusion is arrived at: "The foregoing observations demonstrate that in the dog different kinds of nerve-endings situated in one and the same cutaneous field possess reflex spinal connections differing wholly *inter se*. For discrimination

between certain sets of end-organs in the skin there are in fact available, not only psychological criteria, involving processes of sense, but data purely physiological with characteristics given in tensions of the musculature." SUTHERLAND SIMPSON.

**THE MECHANISM OF THE RESPIRATORY MOVEMENTS OF  
(367) THE GLOTTIS.** FRITZ DE BEULE, *Nervaze*, vol. v. p. 2,  
1903, p. 111.

THE first part of this paper is devoted to an historical account of the views of previous observers with regard to the movements of the glottis, and the part taken by various muscles in producing these. Longet described the posterior crico-arytenoid as the only abductor of the vocal cords, all the other intrinsic muscles being adductors. He regarded the inspiratory opening of the glottis as an active (muscular) movement, and the inspiratory closing as mere elastic recoil. Von Harless, Rhulmann, Ewald, and Jurasz are agreed in considering the lateral crico-arytenoid as a possible abductor. They state that this muscle not only rotates the arytenoid cartilage around its vertical axis, but also draws it slightly forwards, so that a certain relaxation of the vocal cords is produced, and in this way a measure of abduction is said to be brought about. Rosenbach has stated that under certain conditions the crico-thyroid is able to bring about abduction of the cords. Cruveilhier and Meyer credit the inter-arytenoid with being an abductor. They regard this muscle as consisting of two layers of fibres, a shorter attached to the inner margin of the posterior aspect of the arytenoids, and a larger attached to the posterior surface of the cartilage nearer the outer border; to these longer fibres they ascribe the power of rotating the arytenoids outwards around their vertical axes, and so causing abduction of the cords. Beule points out that all these statements are merely hypothetical, and have not been supported by experiment. Krause and Semon, having observed that the opening of the glottis during quiet respiration is always much larger than in the dead subject, came, independently, to the conclusion that the posterior crico-arytenoids (abductors) are in a state of constant tonic contraction, while the adductors remain inactive. In a later paper Semon has given further details. He examined fifty adults; in eight of these the glottis showed movements during quiet respiration; in forty-two no such movements were observed. He regards the quiescent state of the cords as limited to the human subject, stating that in animals constant movements occur. Grossman, having excised the posterior crico-arytenoid, found that respiratory movements of the vocal cords still continued, with only slight

limitation in extent. Kuttner and Katzenstein also found that respiratory movements continued after destruction of the posterior crico-arytenoids. They regard this as incompatible with the theory of constant activity of the abductors and quiescence of the adductors. They incline to the view that there is constant but varying activity of the opposing groups. These authors further state that respiratory movements are the same in man and in dogs, the greater extent of these in animals being due to the excitement and fear caused by the examination. Krause, having divided one recurrent laryngeal nerve, found that the corresponding vocal cord approximated to the mid-line, but did not entirely cease to move with respiration. Grossman found that during the expiratory stage of dyspnoea the crico-thyroid passes into a stage of marked contraction. Krause, in a later paper, claims priority for this observation, and further states that the crico-thyroid contracts during quiet respiration also.

There are, then, two main theories held by various observers with regard to the movements of the glottis :—

(1) That there is active contraction of the abductors during inspiration, and passive recoil during expiration (Longet, Semon).

(2) That there is constant activity of both abductors and adductors, but that this varies in amount to allow of alternate opening and closing of the glottis (Grossman, Kuttner and Katzenstein).

Beule next proceeds to describe the experiments he has performed. These were carried out on dogs. They fall into two main groups. In the first the movements of the vocal cords themselves were watched under various conditions; in the second the contractions of individual muscles were studied.

(1) To obtain a good view of the vocal cords the trachea was opened just below the cricoid cartilage, and a direct view of the glottis obtained from below. Exact measurements of its rima were taken with Exner's laryngometer. On examining in the absence of an anæsthetic, the respiratory movements were seen to be very irregular, the vocal cords separating widely with inspiration as a rule, but also showing smaller oscillations (the rima varying from 2 to 12 mm. in breadth). On expiration the cords approximated exactly. After an injection of morphia respiration became much quieter, and the movements of the glottis less marked. Complete closure still occurred with expiration, while the inspiratory opening varied from 7 to 8 mm. During chloroform anæsthesia respiration was still quieter, and the glottis remained freely open during expiration; respiratory movements were very slight, the cords being frequently quite motionless; the respiratory oscillation was about .5 mm. Dyspnoea was next set up by blocking the trachea, when the movements of the glottis

again became very marked. After death the cadaveric opening of the glottis was found to be much less (3.6 mm.) than during life. Beule concludes from these experiments that the respiratory movements of the glottis are similar in man and in dog, their exaggeration in the latter being due to fear. He further points out that mere recoil after abduction could not carry the vocal cords markedly inside the cadaveric position, so there must be active contraction of the adductor muscles.

Continuing these experiments, the movements of the vocal cords were observed after various intrinsic muscles had been destroyed or their nerve supply cut. The posterior crico-arytenoid was destroyed by dividing the trachea just below the larynx, and drawing this forwards and upward so as to expose the posterior aspect of the cricoid cartilage. The effect of this operation was to limit the amount of movement of the vocal cord of the same side, but not to entirely suspend it until deep anæsthesia was induced. Similarly, after division of the superior and recurrent laryngeal nerves of one side, the vocal cord of that side was not rendered immobile until deep anæsthesia was induced. From these experiments it is evident that there is expiratory contraction of the adductor muscles; that this is of reflex origin is shown by the abolition of the movement by deep narcosis. The movement of abduction in the above cases is due, according to Beule, to a mechanism entirely outside the larynx itself.

(2) To permit of direct examination of the movements of the laryngeal muscles a window was made in the ala of the thyroid cartilage, through which the thyro-arytenoid and lateral crico-arytenoid could be easily seen. On expiration both these muscles, and also the crico-thyroid, were seen to contract vigorously; their movements being exaggerated by the setting up of dyspnœa. The excision of the posterior crico-arytenoid was followed by marked diminution of activity of the thyro-arytenoid and lateral crico-arytenoid muscles, their contractions at the same time becoming irregular. Anæsthesia being induced, the movements of these muscles became at first more regular and then gradually died away. The results here tend to show that all the adductors act together in producing the expiratory narrowing of the glottis.

In a further series of experiments Beule deals with the extra-laryngeal mechanism acting on the glottis. The larynx and trachea having been exposed as before, both superior and recurrent laryngeal nerves were divided; the glottis became immobile, partly open; respiration was hurried and irregular. The central end of one superior laryngeal nerve was then stimulated, expiration became short and strong and was accompanied by retraction of the vocal cords, during which the arytenoid cartilages came

in contact, but the glottis itself always remained partly open. Dyspnoea was then set up by plugging the trachea; at the third respiration the glottis opened with inspiration, returning to its former size at expiration; this enlargement went on progressively, the arytenoids gliding outwards at each inspiration. At the ninth respiration the glottis closed spasmodically and general convulsions supervened. Respiration was then re-established and the wall of the pharynx exposed. It was then seen that with each inspiration the stylopharyngeus contracted strongly, the constrictors of the pharynx remaining passive. On now stimulating the cerebral end of the superior laryngeal nerve the stylopharyngeus was seen to contract more firmly on inspiration, while the constrictors came into play on inspiration. So powerfully did the latter act that the alæ of the thyroid cartilage were markedly approximated, so actually pressing the arytenoid cartilages together. Owing to this action Beule regards the constrictors as accessory muscles of phonation.

To further clear up the action of the stylopharyngeus another experiment was performed. The lateral wall of the pharynx was laid bare, the infra-hyoid muscles cut across near their insertion, the laryngeal nerves divided, and finally the trachea opened in the usual manner. On inspiration the stylopharyngeus was seen to contract vigorously. By its contraction it produces a rocking movement of the hyoid bone upwards and forwards around a pivot formed by the greater cornua, which, in the dog, each consist of three pieces articulating together and fixed to the temporal bone above by a strong ligament. This movement occurs with each inspiration and causes an inspiratory enlargement of the pharynx. When dyspnoea is set up, these movements are exaggerated to such an extent that traction is made on the arytenoid cartilages through the reflection of the pharyngeal mucous membrane on to them, so that they pass slightly outwards, in this way causing separation of the vocal cords to the extent of 3 or 4 mm.

Beule summarises his results as follows:—

- (1) During quiet respiration (obtained by anæsthesia in the dog) the vocal cords are either motionless or show slight oscillation. This is due to a permanent contraction of the abductors.
- (2) During excited respiration the glottis opens widely with inspiration and shuts completely with expiration. This is due to the alternate activity of the abductors and adductors.
- (3) During extreme respiratory efforts (dyspnoea) the stylopharyngeus, sub-hyoid muscles and constrictors of the pharynx all come into play.

HEWAT FRASER.

**PATHOLOGY.****ON THE PRESENCE OF FOREIGN BODIES (CRYSTALLINE  
(368) BODIES AND MICROBES) IN THE NERVE CELL.**  
MARINESCO, *Presse Méd.*, 26th Aug. 1903.

IN 1887 Sudakiewitsch stated that he had found lepra bacilli in nerve cells, and as the lepra bacillus is non-mobile, he concluded that their inclusion was due to phagocytic action on the part of the nerve cell. This explanation has received support from various authorities, including Metchnikoff.

Metchnikoff, in fact, includes nerve cells in his list of fixed cells, which, by means of their amœboid movements, are able to embrace foreign bodies. Marinesco in this paper deals with the foreign bodies, crystals and microbes, which are found in nerve cells under certain conditions.

With regard to the presence of crystals in these cells he states that in a case of hypertrophic pachymeningitis he found crystals in the cytoplasm of medium and small pyramidal cells of the cortex. He has never seen them in a nucleus.

These crystals are soluble in chloroform, but not soluble in water or in alcohol.

He concludes that they are crystals of hæmatoidin which enter the nerve cell in the state of solution, and afterwards separate as crystals, owing to some unknown favourable conditions within the cell itself.

He then goes on to describe a case of leprosy, in which he found many lepra bacilli in the cells of the posterior root ganglia.

In some of these cells the nucleus, surrounded by some chromophile substance, was seen in the centre of the cell, while in the periphery all the chromophile substance had disappeared. In other cells the nucleus lay at the periphery with some chromophile substance, while the rest of the cell was clear.

This clear part of the cell was occupied by a network, in the meshes of which many lepra bacilli were visible. They could also be seen in the chromophile substance, but less frequently. The author then mentions an interesting relationship between these bacilli and the pigments which are found in these cells.

Three different pigments are found: first, a brownish-green pigment around the nucleus of the small cells, which does not stain; second, a yellowish pigment which is present in much larger granules than the first. These granules are often polygonal. Then there is an ochre-coloured pigment which often occupies most of the cell body of medium and large cells. It is only in cells containing this last-mentioned pigment that bacilli are

found in any numbers. A few are sometimes seen in the cells containing the large granules, but none are found in the small cells containing the brownish-green pigment.

The author suggests that the bacilli feed on this fine granular pigment, and in this he agrees with Babes. A somewhat similar appearance is sometimes seen in nerve cells in pellagra and tabes, but the honeycombed appearance is less evident.

It is interesting to note that the lepra bacilli are rarely seen outside the nerve cells, not even in the cells of the capsule or in the small vessels.

As regards the mode of entry of the bacilli into the nerve cells, the author refers to the non-mobility of the lepra bacillus, and denies any amoeboid movement on the part of the processes of the cell. He suggests that they are carried into the interior of the nerve cell by the lymph current.

The work of Holmgren and others has proved the presence of a network of lymph canaliculi in the cytoplasm of the nerve cell, and these intracellular canaliculi are continuous outside with those of neighbouring cells, and with the lymphatic spaces of the nervous system (Pugnat).

The lepra bacilli, then, enter the superficial vessels of a nerve-trunk, and are carried upwards to the posterior root ganglia and anterior cornua, and into the nerve cell, by the lymph stream.

R. G. Rows.

### CLINICAL NEUROLOGY.

**NEUROFIBROSARCOMA.** R. CESTAN, *Revue Neurol.*, Aug. 15, (369) 1903.

THE clinical history of these cases of generalised sarcoma of the cerebro-spinal axis is characterised by similarity in the evolution of the symptoms; in the presence of general signs, headache, vomiting, and optic neuritis; in the early localisation in certain cranial nerves, particularly the auditory; and, finally, in the frequent absence of symptoms pointing to the involvement of the spinal roots or peripheral nerves.

In the case cited, the patient, aged 20, first suffered from left facial neuralgia and paralysis, and left deafness. Two years later the deafness had become complete in the left, and slight in the right ear. Within four years of onset there was complete deafness, bilateral facial palsy, with R.D.; blindness, with optic neuritis and reeling gait; and death followed with the accession of vomiting, tachycardia, and difficulty in deglutition.

Autopsy showed tumours of the eighth, third, fourth, seventh, and tenth pairs of nerves, the spinal roots, and the peripheral nerves. Microscopically the growths were shown to be polymorphic

sarcomata, varying in structure according to location. The foliating character of the growth accounted for the late implication of the axis cylinders in the spinal and peripheral nerves.

The disease is closely related to Recklinghausen's, the differences in characteristic symptoms and location and structure being bridged over by already reported cases.

G. W. HOWLAND.

**A STUDY ON TUBERCULAR POLYNEURITIS.** COLELLA, *Ann. (370) di Neurolog.*, Fasc. ii., 1903.

AFTER a long review of the literature already published on tubercular polyneuritis, and a description of the methods of his research, the author gives a full history of three cases which have recently come under his observation.

This history consists of a very thorough clinical and histopathological examination.

The three cases which he describes illustrate the three types of the disease which are commonly met with.

The first case was an example of what he terms "amyotrophic neuritis." In this case paralysis and atrophy of muscles were the essential features, and disturbances of sensation were very slight.

In the second case disturbances of sensation were more prominent than motor disorders. Pain, anæsthesias and analgesias were the first symptoms of the disease, and throughout its course they were the most prominent. To this condition he applied the name "painful or anæsthetic neuritis."

In the third case pain, anæsthesias and paræsthesias, and delayed transmission of impressions, were associated with motor troubles.

This is an example of "sensory-motor neuritis," and is the type most frequently met with.

He also refers to a class of cases in which no morbid sign was discovered during life, but subsequent examination revealed considerable degenerative changes in the peripheral nerves.

In some cases, and especially in those which give a history of alcohol, the onset is acute; but generally the disease begins insidiously and progresses slowly.

The paralysis, when established, varies in its intensity, its form, and its diffusion. It may be seen as a simple muscular weakness, or it may approach a paraplegic type. The lower limbs are more frequently affected than the upper. Cranial nerves, the vagus, phrenic and facial, may be affected; the ocular muscles and sphincters escape. Atrophy follows the paralysis, and is accompanied by modifications of electrical reactions.

Sensory disturbances are, however, more frequently seen than motor disorders, and they are generally diffuse. Of these,



subjective disturbances are the more marked. Objective signs, such as tenderness of muscles and nerves, pain on movement, and with tension, are less frequent and less constant.

Besides the hyperæsthesias, hypoæsthesias and anæsthesias are sometimes found. They are rarely diffuse. Sometimes also delayed transmission of impressions, and weakening of muscular sense, and of thermic sensibility, are present.

Disturbances of cranial nerves are rare.

Cutaneous reflexes are generally diminished; tendinous reflexes are weakened or may be absent.

Worthy of note is the rapidity with which the reflex power becomes exhausted.

Trophic changes occur frequently, and are seen most in the lower extremities. Hypertrophic osteo-arthropathies have also been described. Vasomotor and secretory disturbances are not rare.

Mental disturbances are somewhat rare, but when present they give rise to a form of mental disease which has been called "polyneuritic psychosis" or "toxæmic psychic cerebropathy." They may appear in the early stages of the affection and may include simple modifications of the character with weakness of memory; or they may show themselves in delirium and partial amnesia. Rarely do they go on to dementia.

The author has previously dealt with these mental disturbances in his monograph, "*Lá Psichici polineuritica*." In this he suggested that, as in other infectious diseases, certain toxines attack the nervous structures—in one case, the peripheral trunks, producing a polyneuritis, and in another case, the cerebral substance, producing psychic symptoms. As an anatomical basis of the latter, he said he thought it probable that the psychic disorders are due to alterations of the nerve fibres which form the systems of associational fibres of the cerebral cortex.

With regard to the pathological anatomy of these cases, the author found that in the case of amyotrophic neuritis, there was a Wallerian degeneration of the intra-muscular nerve fibres, which extended only a little way up the nerve trunks. There was no sign of inflammatory reaction.

He found also a central chromatolysis, with displacement of the nucleus to the periphery in some of the cells of the anterior cornua.

The cell processes in the majority of the cases were not broken.

The muscular fibres were many of them in a condition of atrophy or granular degeneration.

In the second case similar changes were seen in the distal portions of the cutaneous branches of the spinal nerves. The motor nerve fibres and the muscular fibres were not much altered. The third case showed some lesions in the motor and sensory nerves, and in some of the cells of the anterior cornua.

The meninges were not affected at all.

The author then proceeded to discuss the relation between the lesion in the nerve fibres and the lesion in the cells of the anterior cornua.

He first mentioned two theories, each of which has had numerous supporters. The first theory suggests that the lesion of the nerve fibre is a primary lesion, and the second that the lesion of the nerve fibre is secondary to a nerve cell lesion, and this, according to Raymond, may be either dynamic or organic.

He then referred to the experiments which have shown that wounding a nerve fibre produces a lesion in its nerve cell. This lesion is similar to that seen in polyneuritis, and differs only in that it is a much more rapid process.

In both cases the changes in the nerve cell pass through two phases; a first, in which the chromophile elements undergo a dissolution more or less complete, and the nucleus is displaced to the periphery of the cell; and a second phase, in which the fundamental, achromatic substance of the cell undergoes disintegration. The former may be recovered from; the second is irreparable.

In support of the view that the nerve cell changes are secondary, he mentions that they are rudimentary compared with the fibre lesions, and that in some cases they are entirely absent.

But although he adopts the view that the major part of the nerve cell changes are secondary, the author admits that in some cases the same pathogenic influence may act primarily on the nerve cell and on the nerve fibre at the same time.

In his monograph, referred to above, he stated that in infective diseases and in intoxications the neuro-muscular arc may be affected in whole or in any part, thus giving rise to the great variety of morbid pictures seen.

Finally, from the facts recorded, it may be stated that every severe alteration of the nerve cell is followed by a degeneration of the axis cylinder; and from a lesion of the axis cylinder, if it is sufficiently grave, progressive alterations may ensue in the nerve cell.

Moreover, both the nerve cell and the nerve fibre, component parts of one unit, may be injured by the same pathogenic agent at the same time.

R. G. ROWS.

**CONTRIBUTION TO THE STUDY OF HERPES ZOSTER.** ERNST  
(371) HEDINGER, *Deutsche Zeitschrift f. Nervenheilk.*, Bd. 24, H. 3 u. 4,  
1903, S. 305.

THE author describes a case of herpes zoster with autopsy.

The patient, a male, aged 47, was admitted to hospital on

October 16th, 1901, suffering from chronic renal disease and symptoms of cardiac insufficiency; uræmic symptoms developed soon after admission. On November 7th a herpetic eruption appeared in the region of Head's eleventh left dorsal area. On November 25th, nineteen days after the appearance of the herpes, the patient died from uræmia and heart failure. At the autopsy, which was made five hours after death, a marked degree of granular contracted kidney was found with cardiac hypertrophy and dilatation.

The spinal cord and intervertebral ganglia appeared normal to the naked eye. The cord and all the ganglia from the tenth dorsal downwards on both sides were taken out for examination.

With the microscope a large wedge-shaped hæmorrhage was discovered in the eleventh left ganglion. Many of the nerve fibres were destroyed. Other parts of the ganglion showed patches of lymphocyte infiltration, some irregularly scattered, others with a perivascular distribution. There was some increase of fibrous tissue.

The tenth and twelfth left dorsal ganglia showed similar changes, but in a much slighter degree. The lumbar ganglia on the left side also showed some lymphocyte infiltration, but nowhere so pronounced as in the lower dorsal region. In the lumbar ganglia on the right side there was only slight cellular infiltration, the changes being more marked in the eleventh and twelfth dorsal.

The eleventh left intercostal nerve showed a moderate degree of fibre degeneration, but no degeneration was demonstrated in its cutaneous terminations. The nerve branches in the skin showed, however, a very distinct infiltration with uninuclear leucocytes.

A moderate degree of degeneration was present in the posterior root and could be traced into the cord. The degenerated area lay on the mesial side of the left posterior horn, and was traced upwards for three segments. No descending degeneration was demonstrated.

An area of degeneration was also present in the cord lying internal to the pyramidal and direct cerebellar tracts. This degeneration, which was not connected with the posterior roots, the author thinks was probably associated with the renal toxæmia.

Reference is made to Head's observations, which show that the kidney is intimately related with the tenth, to a less degree with the eleventh and twelfth dorsal and first lumbar cutaneous areas. The author is of opinion that the distribution of the herpes in his case is associated with the morbid condition of the kidneys. He argues that there is a group of cases of reflex zoster, the zoster appearing in definite skin areas in consequence of peripheral irritation of sympathetic fibres related to an internal organ. He remarks that further observations are required in order to decide

whether in this reflex form of zoster the changes in the inter-vertebral ganglia described above are always present, also to determine whether discoverable changes are to be found in the peripheral sympathetic fibres, which were not examined in his case.

EDWIN BRAMWELL.

**ON THE HISTOLOGY OF MULTIPLE SCLEROSIS.** The results (372) of investigation by new methods. MAX BIELSCHOWSKY, *Neurol. Centralbl.*, Aug. 16, 1903, p. 770.

In the *Neurologisches Centralblatt* of July 1st, 1902, M. Bielschowsky published the details of a method of impregnating axis cylinders depending on the reduction of an ammoniacal silver nitrate solution by formaldehyde. This method, and a modification of it, which is to be published shortly, have been used by the author in his investigation of five cases of multiple sclerosis, the results of which are given in the present paper.

All five cases were pathologically, and, with one exception, clinically, typical of the disease; one had lasted two years, two were of ten years', and two of twenty years' duration.

Since the silver-impregnation method involved the use of formalin-fixed tissue it was possible to compare neighbouring sections, some prepared by that method, and some stained by other means to show medullary sheaths, etc. Sclerotic patches which, stained by Weigert's method, appeared to be free from fibres, proved to be rich in axis cylinders by the silver treatment. Moreover it was noticeable that both in early and old patches of disease the homogeneous black bands, representing the axis cylinders, were indistinguishable from those found in healthy parts, both as regards their thickness and their regularity of outline. In fact the boundary line between the healthy and diseased parts could often only be made out by the presence of thickened vessels in the sclerotic tissue. On the other hand, in certain patches some of the fibres were more sinuous and varied more in calibre than those seen in normal tissue. In others the changes were still more marked and not infrequently the axis cylinder presented a knobby appearance not unlike a string of pearls, or was split up into a bundle of parallel fibrils, which perhaps united again to form a homogeneous band.

It was interesting to compare the impregnation preparations with those produced by the use of so-called elective axis cylinder stains such as that of Kaplan. By the latter's method a certain cement substance is stained which he has called the axostrome, and which is associated both in its occurrence and in its chemical characters with the medullary sheath. Old sclerotic patches which appeared

fibreless by Weigert's stain and by Kaplan's method often presented a practically normal picture as regards nervous structures in the silver-impregnation preparations. In fresh sclerotic patches, on the other hand, the axostrome stain sometimes portrayed fibres in parts which appeared fibreless by the Weigert method. It would seem, therefore, that one must divide the naked axis cylinders in sclerotic patches into two categories: (1) those in which the axostrome is preserved, and (2) those in which the axostrome is lost. The latter are well seen by the impregnation method, and are, in the majority of cases, not to be distinguished by their appearance from the former or from medullated fibres. As the result of his observations, the author concludes that the large majority of axis cylinders seen in his silver preparations of sclerosis are the persistent remains of the original fibres, but that here and there evidence is afforded of some process of regeneration by the presence of fibres which divide at an acute angle into two branches, one of which may again split up in like manner. He is tempted to regard this sprouting process as one of new formation, and is supported in his view by the fact that similar conditions have been observed in the white matter of embryonic cerebral hemispheres.

M. Bielschowsky claims that by his method the relationship of the clinical symptoms to the anatomical findings is rendered clearer than by any other method, and that the absence of secondary degeneration in those cases where the same fibre system traverses numerous patches of sclerosis is made comprehensible.

In conclusion, he discusses the histogenesis of the disease, and inclines to the view that it is primarily an inflammatory process, and one which attacks both the parenchymatous and interstitial tissues.

E. FARQUHAR BUZZARD.

**MENTAL DISTURBANCES IN A CASE OF DISSEMINATED (373) SCLEROSIS.** M. LANNOIS, *Rev. Neurol.*, Sept. 15, 1903, p. 876.

A CASE of disseminated sclerosis in which, after the patient was more than a year under observation, psychical troubles developed abruptly—delusions of exotic form, ideas of persecution and megalomania. In such cases the pathological condition is probably certain microscopical cortical changes, or a macroscopic lesion with special localisation.

C. MACFIE CAMPBELL.

**SYPHILIS AS A CAUSE OF CHOREA.** L. HARRISON METTLER, (374) *Am. Journ. of Med. Sc.*, Sept. 1903, p. 481.

THE author, in a paper read before the Chicago Medical Society, gives a resumé of the literature regarding syphilis as a cause of chorea, and tries to explain the connection between the two.

He contends that the name *chorea* indicates a symptom rather than a definite disease, and that under it many varied affections are described. He thinks that the distinction drawn between Sydenham's chorea and chorea due to gross organic lesions is artificial, the symptom of chorea pointing simply to some functional disturbance of the higher motor neurons, which may be due to various irritative causes.

He mentions records of six cases of chorea associated with syphilis, and cured of the attack by anti-syphilitic treatment. Of these, four were unilateral, two bilateral. He also gives, *in extenso*, the record of a case of his own, with typical chorea and unequivocal inherited syphilis, in which, after all the usual remedies proved fruitless, the use of iodide of potassium was speedily successful.

The view that Sydenham's chorea is due to a toxic nutritional or molecular change in the cells of the cortex is explained, and the writer considers it only reasonable to suppose that the toxin of syphilis can and does act like any other toxin in producing ordinary chorea.

The paper is summed up by the following conclusions:—

1. Syphilis in rare instances is a cause of chorea.
2. Chorea may be the result of acquired or of hereditary syphilis.
3. Most of the cases of syphilitic chorea are unilateral.
4. Chorea is not a disease, but a mere symptom.
5. These cases should be treated as cases of syphilis, not of chorea.

JOHN D. COMBIE.

**CHOREA AND GRAVES' DISEASE.** G. A. SUTHERLAND, *Brain*, (375) Summer 1903, p. 210.

THE author insists that a closer connection exists between chorea and Graves' disease than is generally supposed. He begins by stating the opinion of Gowers that movements of a choreiform type form a complication, and of Dieulafoy that choreiform movements may form part of the motor troubles associated with Graves' disease, but do not form a true chorea.

He then records two cases in both of which true chorea in childhood preceded the onset of typical attacks of Graves' disease at the ages of 26 and 16 respectively. These controvert the statement of Dieulafoy that cases of Graves' disease have never been recorded as following on chorea.

He then points out that the names *Graves' Disease* and *Chorea*, serve merely to indicate a collection of symptoms. He suggests that the disorders present in the two may not have the same cause, but that they depend upon a similar affection of the central nervous system, which in childhood produces the set of symptoms

known as chorea, and in adult life those known as Graves' disease. He ends by enumerating some dozen clinical features in which the two agree.

JOHN D. COMRIE.

**NOTE ON THE TREATMENT OF CHOREA BY ASPIRIN.** R. T. (376) WILLIAMSON, *Lancet*, August 22, 1903, p. 526.

THIS is a statement of the writer's experience in the treatment of 35 cases of chorea minor by Aspirin, without confinement to bed. The treatment is strongly advocated, merely on empirical grounds, though the writer is compelled by the steady improvement of all his cases, by the fact that several treated for long periods unsuccessfully with other means, improved at once on aspirin, and by the rapid betterment of severe cases, to conclude that a further trial may establish the beneficial action of this drug.

He began with doses of 10 grains thrice daily, increased to 15 grains four times daily, and saw, in all cases, marked improvement in six or seven days. He includes in the article records of eight of the most noteworthy cases.

JOHN D. COMRIE.

**A CASE OF AUTOMATIC WANDERING LASTING FIVE DAYS.** (377) W. S. COLMAN, *Lancet*, August 29, 1903, p. 593.

AUTOMATIC wandering is a rare condition of great interest, both from its neurological and medico-legal aspects. It is met with as a post-epileptic state both after major and minor epileptic attacks. A few cases have, however, been described in which the wandering seems to have replaced an epileptic fit. The case here recorded is in the opinion of the author to be classed in this group.

The patient, a man of 37, came of a neuropathic family. An elder brother and a paternal uncle suffered from epilepsy. Another brother died in infancy of convulsions, and his father died of "softening of the brain." The patient had had two prolonged attacks of automatic wandering, lasting thirty hours and five and a half days, besides many of short duration. He had never had an epileptic fit and there was no definite history of petit mal, although he stated that occasionally when walking about a "brownish mist" came before his eyes. There were no signs of organic disease.

Dr Colman has described two similar cases (*St Thomas's Hospital Reports*, 1898), in which the automatic wandering appears to have replaced an epileptic fit. A brief account of these two cases is given in this paper.

A case of automatic wandering recorded by Charcot is referred

to in which the condition lasted for eight days, and also a case reported by James where the automatism persisted for no less than two months.

EDWIN BRAMWELL.

**ACROCYANOSIS AND WRITERS' CRAMP.** E. BRISSAUD, L. (378) HALLION, H. MEIGE, *Arch. gen. de Méd.*, Sept. 15, 1903.

IN this paper the authors describe and discuss a case in which vaso-motor spasm and writers' cramp occurred together.

The patient was a boy of 16. His physical condition was peculiar, he was vain and always striving to be thought original and clever. He boasted of his abilities as a comedian and as a smoker; at times he spoke slowly and quietly, the next minute he would be shouting. He would do anything to be thought unlike other people. This peculiarity took the most active shape in the method and manner of his writing. (1) His position when writing was always peculiar. (2) When told to write he would begin correctly, but at the end of a line he would say that he could do no more; he would then rest, but on resuming, the next line would be quite illegible. (3) The characteristics in the writing were of two kinds: (a) some letters were quite unformed or reduced to points and accents, chiefly letters which were unadornable; (b) he would replace the usual letters with strange and incoherent characters, especially if he could make something very original. (4) When interested in his subject he would write quite well.

This condition the authors regard as "functional" and sum up thus:—He could not write, except when he was interested in the actual thing he was writing, without thinking: (1) that he was writing; (2) that he could not write.

The second point in the case was that his hands had always been blue, cold and numb, and had an awkward feeling. If the skin were pressed the blanching which resulted remained for a long time. Plethysmographic tracings were taken, but no capillary pulsation was recorded nor could any difference in the volume of the fingers either when the hand was raised above his head or hung down at his side be detected. When, however, the hand was placed in water at a temperature of 43°-44° Centigrade, capillary pulsation was recorded and normal differences in the volume of the fingers depending on position were obtained. This result lasted for some considerable time after the hand was removed from the water, and the bleaching which was caused by pressure on the skin was only very transitory.

Whether the vaso-motor spasm had any influence on the motor condition could not be ascertained, as the patient refused to be treated regularly. The paper concludes with arguments as to the



cause of the condition : (1) Did the vaso-motor condition cause the disorder of the motor function? (2) Did the motor disturbance cause the vaso-motor spasm? (3) Do both conditions depend on the same cause?

They conclude that the motor disorder and the vaso-motor phenomenon as well as the peculiar psychical disturbance are all due to one and the same cause—a cortical affection.

T. GRAINGER STEWART.

**A CONTRIBUTION TO THE PATHOLOGY OF PARAMYO-**  
**(379) CLONUS MULTIPLEX (FRIEDREICH'S TYPE).** J. RAM-  
SAY HUNT, *Journ. Nerv. and Ment. Dis.*, July 1903.

THE author, after giving a brief review of the literature of the condition, mentions the diversity of opinion as regards both the classification and pathogenesis of the condition.

The theories regarding the origin of the condition which at present receive serious attention are two in number, the one attributing it to cerebral, the other to spinal origin.

In favour of the spinal theory he mentions the fact that electrical stimulation of the motor cortex with the most delicate electrode fails to produce a contraction of an individual muscle, the result being a movement, a synergetic muscular action. The medullary centres, however, have a more individual relation with the respective muscles under their control, and he mentions that Sano advocated an individual muscle representation in the anterior horn cells based on experimental studies. The rare cases in which paramyoclonic twitchings occurred in the course of organic disease of the cerebral cortex, such as chronic meningitis with cortical atrophy (Murri), chronic uræmic œdema of the cortex (Levi and Follet), and myoclonus epilepsy (Unverricht, Clark and Prout), have been considered confirmative of a cortical origin by some observers. The author points out, however, that if minute pathological changes of the cerebral cortex are capable of inducing this type of muscular contraction, cases should be of more frequent occurrence, and that in view of this rarity a coexisting alteration of the spinal centres is more probable.

The family affection characterised by the association of myoclonus and epilepsy is one in which there is a degenerative process of the cerebro-spinal axis, the epilepsy being referable to the upper, the myoclonus to the lower centres.

In favour of the spinal theory he advances those cases of paramyoclonus multiplex occurring with corticular affections. Chauffard's case of hemiparamyoclonus, which followed directly on an attempt to break down the joint adhesions in the right knee and

hip, and also a case of spondylose rhizometique with the usual joint affections reported by Levi and Follet, which was complicated by paramyoclonus.

The articular muscular dystrophies, and the old theory that they depended on perversion of the functions of the anterior horn cells, is brought forward in this connection, and he mentions that Raymond demonstrated experimentally that if the posterior roots are divided before the articulation is disturbed the atrophy will not occur. He then urges that if pathological reflex stimuli starting from the articulations can affect the trophic function of the anterior horn cells, it could not be unreasonable to infer that the motor function may be perverted in a similar manner with a resulting hyper-excitability. The fact that the tendon reflexes are exaggerated in muscles where this atrophy has occurred is well known, and he cites a case reported by Sir William Gowers in this connection.

The muscles from a case of articular atrophy were examined by Darkschewitz, who found that the muscle fibres had undergone a diminution in size averaging about half the normal diameter. These changes occurring in a case where the trophic functions of the anterior horn cells had been restricted by some inhibitory influence, have all additional significance when the muscle changes found in his case of paramyoclonus multiplex are considered. He also brings forward as further evidence in favour of the spinal theory the close relationship between the contractions of paramyoclonus multiplex, myokymia, and fibrillary twitchings.

The author next gives a clinical account of a case of paramyoclonus which occurred in a man aged 49, who was suffering from a tubercular ankle for which he was in hospital when the myoclonic condition was observed. He died from acute tuberculosis. On histological examination the nervous system was entirely normal. The muscle fibres, while retaining their normal structure, were considerably hypertrophied. This increase in size of the fibres was not distributed universally over the field, but about two-thirds of the fibres showed this change. The sarcolemma nuclei were often seen scattered between the sarcous elements.

T. GRAINGER STEWART.

**A CASE OF INCREASED PITCH OF VOICE IMMEDIATELY  
(380) FOLLOWING CEREBRAL HEMIPLEGIA** (Paralysis of the  
Cricothyroid of Central Origin). J. GRASSET. *Rev. Neurol.*,  
Sept. 15, 1903, p. 873.

THE patient, a woman of 70 years of age, had a typical left-sided hemiplegia, the face being slightly involved. Immediately after

the attack it was noticed that the pitch of her voice was raised (voix eunucoïde), the patient being unable to utter deep notes. No loss of voice nor difficulty of articulation, no hoarseness, no aphasia, no dysphagia nor respiratory trouble. Hysteria could be eliminated. The vocal cords were normal: the cords came together well. When patient tried to utter deep notes, the glottis became irregular, due to want of tension of the vocal cords. No unilateral paralysis of the thyro-arytenoid.

If one fixed the thyroid cartilage the voice resumed its natural pitch previous to the attack. G. supposes that the condition is due to the paralysis of the crico-thyroid muscle, which results in the thyroid cartilage being approached to the arytenoids on the contraction of the thyro-arytenoids, thus shortening the length of the vocal cords.

C. MACFIE CAMPBELL.

**ON THE MEASUREMENT OF MUSCULAR TONUS.** G. CON-  
(381) STENSOUX and A. ZIMMERN, *Rev. Neurol.*, Sept. 15, 1903,  
p. 881.

THE authors consider that we may take as measure of the muscular tonus the number of stimuli per second necessary to produce tetanus of the muscles. For a normal muscle the number is about 17. In a series of cases examined, where the muscles were clinically recognised to be in a state of hypotonicity, as in several tabetics and hemiplegics, the number varied from 22 to 28. In three cases of increased muscular tonus (two cases of familial paraplegia, one of transverse myelitis) the number was 16.

C. MACFIE CAMPBELL.

**A CONTRIBUTION TO THE STUDY OF SENSORY SPINAL**  
(382) **LOCALISATION.** J. DEJERINE, *Journ. de Phys. et de Path.*  
*gén.*, No. 4, July 1903, p. 656, with 1 Pl. and 2 Figs.

IN syringomyelia and hæmatomyelia the lesion of the posterior horns of the cord causes sensory troubles, with a distribution corresponding in a general manner to the distribution of the posterior roots. The lesion is usually so extensive that it is difficult to say what level of the posterior horn corresponds to the cutaneous distribution of a definite root. The case which Dejerine publishes, with the description of the cord lesions, shows that each posterior root terminates in the corresponding posterior horn at a level corresponding to the point of entrance of the fibres of the posterior root.

The patient, after a fracture of the spine, had a complete paraplegia, both motor and sensory; in addition, on the right side

of the trunk and arm, he had analgesia and thermoanæsthesia with conservation of sense of touch (syringomyelic dissociation), over an area corresponding to the distribution of the 8th to the 12th dorsal nerves inclusively, also of the 1st and 2nd dorsal, and 7th and 8th cervical nerves. The paraplegia was explained by the traumatic lesion of the lumbar cord. To explain the dissociation symptoms there was found a cavity on the right side reaching up to the 7th cervical pair; this cavity separated the anterior horn from the posterior, at the same time dividing the posterior horn in all its length up to the substantia gelatinosa Rolandi into two parts. The lesion in the posterior horn had involved all the elements, while the parts of the posterior roots which were extramedullary and within the column of Burdach were intact. The lesion had attacked that part of the grey matter where the major part of the fibres of the posterior roots end, and had caused the dissociation symptom in the sensory distribution of the corresponding posterior roots; the distribution was as accurate as if it had been a root lesion.

The case shows again that it is in the grey substance at the base of the posterior horns that heat and pain are conducted—not by Gowers' tract (v. Gehuchten, Brissaud). The grey column of the posterior horns may be looked upon as composed of a series of segments, each one of which corresponds to a posterior nerve root. A sensory metamerism in the cord exists no more than a motor metamerism.

C. MACFIE CAMPBELL.

**THE PHENOMENON OF BABINSKI IN THE INSANE.** By (383) EDOUARDO AUDENINO, *Giorn. d. R. Acad. di Med. di Torino*, Luglio-Augosto 1903, p. 465.

THE writer has examined 319 insane patients, excluding all cases where an organic hemiplegia existed.

Among 187 males he found—

Plantar reflex absent in 61 cases,	.	.	32·6 per cent.
Normal on both sides in 78 cases,	.	.	41·7 „
Normal on one side, absent upon the other, in			
25 cases,	.	.	13·3 „
Babinski's reflex upon one or both sides in 23 cases,	.	.	12·2 „

Among 132 women he found—

Reflex absent in 33 cases,	.	.	25·0 „
Normal on one side, absent upon the other, in			
14 cases,	.	.	10·6 „
Normal on both sides in 65 cases,	.	.	49·2 „
Babinski's reflex in 21 cases,	.	.	9·09 „

He has also examined 120 healthy young adults and found—

Reflexes absent in 37 cases,	.	.	.	30·8 per cent.
Absent upon one side in 11 cases,	.	.	.	9·1 "
Normal on both sides in 47 cases,	.	.	.	39·1 "
Babinski's reflex in 25 cases,	.	.	.	20·8 "

A case of hysteria in which Babinski's reflex was constantly present is reported, as is also a case of amyotrophic lateral sclerosis, in which the phenomenon was not present. There was no pathological examination in either case.

JAMES COLLIER.

### PSYCHIATRY.

#### A CONTRIBUTION TO THE ETIOLOGY OF MENTAL DISEASES.

(384) TH. TILING, *Centralbl. f. Nervenheilk. und Psychat.*, Sept. 1903.

MENTAL diseases may be well defined as unusual modifications of normal mental life. Consequently clinical psychiatry should be treated in close union with psychology, to which it is really a supplement. Psychology does not, however, proceed from the same standpoint as psychiatry, so that for the present this union seems impossible. Individual psychology concerns itself mainly with the elements of mental function, or more accurately, with measurements of the components of mental function, and therefore cannot serve as a basis for practical psychiatry. Psychology must, however, in the future practise the synthetic method, even as psychiatry does with its classification of psychoses; and attempt a division of normal individuals through observation and description of the whole man and not of the intellect alone. The ætiology of psychoses will then lead ever into the region of normal psychology, and the transition of normal into morbid mentality be made manifest. It will certainly prove that there exists in mental disease a want of harmony between the elements of the mental organism; certain parts preponderating at the expense of others. If it should be objected that disease, above all, means anatomical changes which the chief object of psychiatry is to investigate and depict, it should be remembered that the anatomical structure and its pathological changes do not occasion an action, but can only influence or disturb it in deeply lying paths, and that mental activity carries in itself its laws, following in detail physiological or psychological law. Now the foundation of human mental life is the emotional sphere, and exists throughout the whole life in the most intimate dependence upon affective states, but this sphere escapes, so far as it is not motorially expressed, the investigation of experimental psychology. Only with the highest culture is there remarked an emancipation of the

intellect, a regulation of, or relative dominance over, the affections. Even this relative dominance, however, is far from being a common property of civilised society. The majority are unable of themselves to analyse their sympathies and antipathies, to separate their ideas of right and wrong from the influence of primitive affective states, and accept their ethical views second-hand, and but half understood, from their leading national spirits. Further there exists in the majority a profound disharmony between these acquired higher concepts—such as righteousness, equity and humanity—and their lower impulses, passions and emotions which are the mainspring of conduct, though of this disharmony they are frequently unconscious. In the great minds of the age, on the other hand, there has been a fortunate constellation of their faculties of feeling and intellect, and in the harmony and not the strength of affective and intellectual states lies the secret of their greatness. Isolated gifts and talents guarantee no certain fruition, but evidence a lack of equilibrium portentous of disaster. It is, however, in the defective elements of society that this disharmony is most evident, resulting in an utter inability in many to comprehend concepts or ideals, influencing minds more happily constituted and co-ordinated. It is true that in the public eye the liar, the dishonest, and the malefactor possess a complete recognition of the moral law, yet to the judge the mind of the judged is a *terra incognita*. To the criminal, truth and honour are words without meaning and have a different interpretation to that of others, though he may have a code of honour and of pride, just as he has a jargon, of his own, unintelligible to others. So with the insane this disharmony exists, and is to be approached by a study of the components of character, particularly in the sphere of the emotions of normal individuals. The old division into four temperaments is now quite insufficient, but it was right in principle and capable of development. For the author, temperament expresses the *tempo* according to which the individuals react to impressions, sensorial or memorial, and may be powerful or feeble, lively or slow, deep or superficial, and lasting or transitory. This gives the form of reaction, which added to the content, gives the specific reaction. Normal people classified according to their specific reactions would fall under one or other of a series of categories of character, to each of which there would pertain its appropriate pathological state, the gradations between the normal and pathological merely awaiting observation to be verified. It would appear indeed that given a character of a certain type, its development into complete alienation becomes inevitable. A character depressed, anxious, brooding over the discomfort of things, seeks for some explanation, some solution of a disharmony, felt but not understood. A true solution, seeing that this disharmony rests on a permanent fundamental

constitution of the elements of the character, is of course impossible, but as the discomfort becomes more urgent, ideas, fragments of earlier reflection, and also sudden new thoughts incongruous with reality, present themselves. Certainty, even an evil certainty, is preferable to uncertainty. An idea is grasped which to the patient affords an explanation and is received as an inspiration. The particular idea is seized to the exclusion of others, because it is a presentation in harmony with the patient's inherent affective state. This idea is not only believed by the patient to be of great value, but it works in him a veritable revolution, placing the whole world in a new light, just as in cases of religious conversion, to which it is indeed akin. Viewed from the point of view of alienists, this is the turning-point in the scale, and this union of idea and ego it is which distinguishes a delusion from a merely erroneous opinion.

It is impossible in this brief abstract to follow the author in his application of this theory to the various psychoses, and which throughout is of great suggestive interest.

R. CUNYNGHAM BROWN.

**ON DEMENTIA PRÆCOX.** ALEXANDER BERNSTEIN, *Allg. Zeitschr.* (385) *f. Psychiatrie*, Bd. 60, p. 554.

THE author objects to the name "dementia præcox," because it is used in various senses by different writers (especially Russian), and because other forms of mental disease can correctly be said to lead to a premature dementia. A new name is therefore to be desired, and it ought to be one that recognises the special motor symptoms as constituting an essential part of the clinical picture. The state of tension in the muscles he would not call catatonic, but rather "paratonic," and he applies the same term to the insanity, which accordingly is named "dementia paratonica progressiva," or, more shortly, "paratonia progressiva." The stupor and muscular elasticity in this disease are quite different from the similar symptoms in circular insanity. The movements have the character of gymnastics; the *negativismus* is the resistance of a gymnastic apparatus. The idiomuscular contractions upon slight mechanical irritation are to be attributed to an autointoxication of sexual origin.

The reporter can fully agree with Bernstein and other authors that the term "dementia præcox" is a very unfortunate one. R. Sommer has recently ("Zur Kritik der Dementia præcox," *Beiträge zur psychiatrischen Klinik*, 1903, p. 184) advocated the use of the name "primary dementia" (Rieger), as expressing the absence of every exterior cause. But we don't know anything

at all about the etiology, and the reporter has elsewhere pointed out reasons for avoiding any designation of the disease based upon the term dementia. As to "paratonia progressiva," the course of the disease is too irregular to warrant its inclusion in the name. There is, moreover, no real progression of what Bernstein calls the paratonic movements. In many of the cases the muscular symptoms are even completely wanting, and most authors are still inclined to exaggerate their importance.

A subsidiary proposal of the author to adopt the name "morbus Kraepelini" can scarcely be taken seriously into consideration. Kraepelin has here continued the work of Kahlbaum, whose highest merit has not been to bring into relief a series of symptomatological peculiarities, as Bernstein seems to assume, but to indicate the principles of the method of clinical research in psychiatry.

HANS EVENSEN.

**A NOTE ON PERIODIC INSANITIES, WITH REPORT OF THREE (386) CASES OF INTERMITTENT MELANCHOLIA.** ALFRED GORDON, *Journ. Nerv. and Ment. Dis.*, Sept. 1903, p. 558.

THE periodic psychoses known are mania, melancholia and paranoia. Krafft-Ebing and Kraepelin also include the circular insanities.

Melancholia is less often periodic than the other forms, but the author maintains that it occurs more often periodically than is supposed, its periodicity being often overlooked owing to the patient passing out of observation.

In the periodic forms of insanity there is an unusually pronounced degenerative basis, and this factor is well marked in intermittent melancholia.

From a prognostic point of view it is important to differentiate between ordinary melancholia and intermittent melancholia. The former is, generally speaking, a curable affection; on the other hand, a patient suffering from the latter affection may recover from an individual attack, but the tendency to a repetition will always be present, and each subsequent attack is liable to be more serious and of longer duration than the one preceding.

Ordinary melancholia is insidious in its onset, and disappears gradually and very slowly. Intermittent melancholia is sudden in its onset and equally abrupt in its termination. The duration of the attacks, as well as of the intervals between the attacks, presents no characteristic features. There is comparative mental lucidity between the attacks, which is rather an unexpected phenomenon in view of their repetition.

The author had the following three cases under his observation for four years:—



CASE I. A woman aged 35, after her fourth confinement and while in apparently perfect health, became suddenly depressed and felt she could not do her work. Conscious of this condition, the patient soon reached a state of complete discouragement, and ceased to take an interest in her household, husband and children. She had perversion of taste and smell, and rejected her food with disgust. She lost weight. Precordial pain was frequent. A blood examination showed a leucocytosis of 22,000 per c.cm. She had headache, insomnia, dreams, and occasional visual hallucinations. The patient remained in this condition two months, and then awoke one morning calm and peaceful, asked for her children, and began to take an interest in all her former duties. The mental faculties became clear and normal.

She remained well for ten months. Three weeks after her fifth confinement the old mental trouble returned suddenly and without any preliminary symptom. This second attack was more severe, and the patient was suicidal. The condition lasted two months, and terminated abruptly like the first. A third pregnancy occurred, and six weeks after confinement she again had an attack of melancholia, which ensued unexpectedly, lasted four months, and again ended abruptly. The patient has now been well for a year. She is not pregnant. Of her eight children, two died with hydrocephalus, a third immediately after birth, and a fourth from diphtheria. Patient's sister and grandmother were insane.

CASE II. A married woman, aged 31, had three attacks. The first was mild in character; the second began suddenly without any preceding physical indisposition or mental disturbance, the patient waking one morning, after having spent the previous night at the theatre, in a state of mental depression accompanied (as on her previous mild attack) with a facial neuralgia. The neuralgia lasted one day. The melancholia was characterised by delusions of unworthiness, visual and aural hallucinations and an array of neurasthenic symptoms. This state lasted three months and ended abruptly.

Six months later she had her third attack, which was of a more severe nature, and lasted six months. In its mode of origin and termination it resembled the former attacks.

All the patient's sisters were of neurotic temperament; an uncle died of parietic dementia, and a grandmother died in an asylum. The patient herself at fifteen had two attacks of chorea in the same year.

CASE III. A married woman, aged 34, during convalescence after an attack of phlebitis, following on confinement, suddenly became depressed—a simple melancholia without delusions. This

first attack lasted six months and terminated abruptly in recovery.

A second attack suddenly occurred six months later, lasted three months, and again the patient recovered in the same rapid manner. During the next three years the patient had two further attacks resembling the former ones, only the symptoms were more intensified, numerous delusions being present which led to attempts at suicide. This third attack lasted six months and terminated in recovery in the same manner as before. She remained in good health for eight months, when the fourth attack ensued with numerous delusions, refusal of food, and occasional auditory hallucinations. This attack has lasted over a year, and there are no signs of improvement.

The patient's three sisters were twice confined in an asylum; an uncle on her father's side is a paranoiac, and the patient herself at the age of ten had epileptiform convulsions.

H. DE M. ALEXANDER.

### TREATMENT.

**THE SURGICAL TREATMENT OF FACIAL NEURALGIA.** A. (387) VAN GEHUCHTEN, *Névralgie*, vol. v. f. 2, 1903, p. 199.

ON the basis that the essential lesion in trigeminal neuralgia is located in the Gasserian ganglion, radical treatment is to be directed either towards the removal of the ganglion itself or to the interruption or destruction of the fibres passing from the ganglion to the cerebro-spinal axis. The division or resection of the nerve trunks on the peripheral side of the ganglion, although followed by a degree of chromolysis in the ganglion cells, does not lead to their permanent degeneration, hence the possibility of regeneration of the nerve-fibres and recurrence of the neuralgia. On the other hand, if the sensory root of the fifth nerve be divided on the cerebral side of the ganglion, all the sensory fibres of the bulbo-spinal root undergo complete degeneration, a result which is equivalent to removal of the ganglion itself. Division of the fifth root above the ganglion is not only an easier operation than that of removing the latter, but has the advantage that the connections of the ganglion with the periphery are left intact, whereby the distressing ocular complications are prevented. While this operation has been completely successful in the hands of Frazier, Keen and others, it is still a formidable undertaking, only a little less so than removal of the ganglion, and Van Gehuchten proposes to bring about a similar degeneration in the fibres of the sensory root by less severe measures. As a result of experimental work

on the vagus, sciatic and other nerves, he believes that the violent and sudden avulsion of the peripheral trunks of the fifth would suffice to bring about such a degeneration both in the nerve-cells of the ganglion and in the fibres of the sensory root between the ganglion and the brain. On theoretical and experimental grounds, therefore, he recommends the trial of the avulsion method before having recourse to the more formidable intracranial operations.

ALEXIS THOMSON.

**A CASE OF LARGE BLOOD CYST IN THE ARACHNOID SPACE  
(388) SIMULATING BRAIN TUMOUR; OPERATION; RECOVERY.**

JAMES TAYLOR and CHARLES A. BALLANCE, *Lancet*, August 29, 1903, p. 597.

THE case here recorded is that of a man aged 34, who, *three months* after a fall on the right side of his head, which kept him off work for one day, began to suffer from right-sided headache. Later, optic neuritis, mental dulness, a variable degree of left hemiplegia, and other symptoms suggestive of a cerebral tumour developed, the available data pointing to the right frontal region as the most probable seat of the lesion. The symptoms increased, and potassium iodide in large doses failed to produce improvement. An operation was decided upon. The operation was performed in three stages. On September 1st a large area of bone was removed. Five days later the dura was opened, and a tense, fluctuating, pulsating swelling of a maroon red colour exposed. The pulsation was expansile. More bone was removed in order to expose the whole extent of the tumour. On September 18th a temporary ligature was placed on the internal carotid, and the tumour, which proved to be a large blood cyst, was removed. The cyst was not adherent to the brain, but was connected with the cerebral arachnoid over the inferior aspect of the temporo-sphenoidal lobe by a slight vascular adhesion. It was removed with little bleeding. The cyst, which measured seven inches in length, four inches from above down, and one and a half inches in thickness, had produced considerable flattening of the convolutions on the right hemisphere. The patient made a good and complete recovery.

EDWIN BRAMWELL.

**THE ELECTRICAL TREATMENT OF HEMIPLEGIA, ORGANIC,  
(389) HYSTERICAL AND HYSTERO-ORGANIC. A. LAQUERRIÈRE  
and LOUIS DELHERM, *Arch. d'electr. méd.*, No. 127, 15 July  
1903.**

THE authors point out that the various works on electrotherapy fail to distinguish sufficiently between the three varieties of

hemiplegia mentioned. The numerous cases of organic hemiplegia reported as cured by static electricity were probably cases of hysterical hemiplegia. The reported cases of improvement under galvanism make the supposition of the presence of an hysterical element not unlikely; similarly one cannot accept without criticism the results after treatment with faradism.

Their own observations lead the authors to advocate the use of galvanism in organic hemiplegia to counteract the muscular wasting and vaso-motor changes. If contracture be present, galvanism is still useful, but it is useless to daily tetanise the muscles with the faradic current. Static electricity is here of doubtful utility and the arterial tension needs to be watched.

In hysterical hemiplegia all the forms of electricity are of use, but especially the faradic current; where hysteria is added to an organic hemiplegia, faradisation may eliminate the functional element while galvanisation is indicated as treatment for the organic affection.

C. MACFIE CAMPBELL.

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## Review

### STUDI CLINICI ED ANATOMO-PATOLOGICI SULL' IDIOZIA

G. B. PELLIZZLI. Torino, Tipografia A. Spandre e C., 1903, pp. 144. Two plates.

THIS work consists of four papers, which appear in the contemporary volume of the *Annali di Freniatria*. It forms the second series of the author's published researches upon idiocy. The first series, which was published in the same journal from 1899 to 1901, dealt with the subjects of idiocy associated with tuberculous sclerosis, the classification of the various forms of idiocy, and the relation of idiocy to epilepsy. The present series is chiefly concerned with microgyria, microcephaly, hydrocephalus, anencephaly, and the general pathogenesis of idiocy and imbecility.

In the first paper there is described a case of microgyria in a boy of seven, who presented also the features of Little's disease. The convolutional anomaly affected the outer aspect of both hemispheres, excepting the occipital lobes. The chief microscopical changes found in the cortex consisted in great diminution in the number of the small pyramidal cells, and rarefaction and decrease in the size of the large and medium pyramidal cells. The author adopts the distinction, recently introduced by Bresler, of micro-

gyria and ulegyria. In microgyria proper, to which category the author's case belonged, there is a subdivision of the convolutions into numerous small gyri in which the cortical strata are continued without interruption. The convolutions may be slightly diminished in size, or they may have normal dimensions. The surface is smooth. The evidence of previous pathological processes is extremely slight. In ulegyria the size of each convolution is distinctly diminished and the sulci are wide. There is interruption of the cortical strata. There is distinct evidence of antecedent pathological processes in the occurrence of roughness and erosions of the surface and cicatricial indurations. Whilst he recognises that this is a correct distinction, Pellizzi considers that it is not one that can always be made, as there are transition forms having the characters of both groups. Moreover, they are not essentially different in their pathogenesis. According to the most authoritative opinions on the subject, both are to be attributed to a meningitis or meningo-encephalitis occurring generally during foetal life. In ulegyria the abnormalities are more marked, the convolutional alterations being constantly accompanied by heterotopia and frequently by porencephaly and defects of the corpus callosum; but they pass gradually into those of microgyria. The author dismisses as untenable the view that microgyria and ulegyria are simply developmental anomalies. They are entirely different in character from the abnormalities in tuberous sclerosis, which there are satisfactory grounds for attributing to a simple developmental error, and they are such as would naturally result from certain inflammatory lesions occurring at an early period of development. The author's case was clinically complicated by Little's disease, and, *post-mortem*, there was found to be an arrested development of the pyramidal tracts and diminution in the number of cells in the anterior horns of the spinal cord. Many authorities would now confine the term "Little's disease" to those cases in which there is spinal spastic rigidity in individuals born before term, and would regard as distinct from it cases of infantile cerebral diplegia. The former they explain as the result of developmental arrest of the spinal portion of the pyramidal tract, the latter as the consequence of meningitis, encephalitis or traumatism. Pellizzi, however, in agreement with some other observers, contends that these two forms cannot be entirely separated from each other, and that the developmental origin of the first is by no means certain, as there are experimental observations which show the possibility of primary degeneration of the spinal pyramidal tract being of toxic origin. On these grounds the occurrence of Little's disease in this case does not necessarily give support to the theory of the purely developmental origin of microgyria, which, moreover, is only rarely found in Little's disease.

The congenital spastic rigidity which is so frequently a clinical symptom in cases of microgyria, he explains upon the hypothesis of the existence of two sets of fibres passing from the cerebral cortex to the cells of the anterior horns of the spinal cord, the one excitatory and the other inhibitory, and anatomically separate in both their cerebral and spinal courses, and therefore capable of being affected singly. He notes that it has been observed that in those cases in which the alterations are limited to the pyramidal cells, the polymorphous cells being apparently normal, there is always spastic rigidity.

The second paper is upon heredity and certain clinical symptoms in their bearing upon the question of the pathogenesis of mental deficiency. The author refers at the outset to Giacomini's classical work upon microcephaly, in which two forms of this condition are distinguished, namely, true microcephaly, the manifestation of a developmental anomaly; and microcephaly accompanied or exclusively determined by a pathological process. Later writers, including the author, have endeavoured to apply this fundamental biological distinction to all forms of idiocy and to classify them in three groups, according as they are of developmental, pathological or mixed origin. Tanzi and others have attempted to define the distinctive clinical characteristics of the developmental and pathological groups. It has been maintained that the common form of idiocy and imbecility (the developmental), is distinguished by an absence of epilepsy, paralysis, paresis, contracture, etc., and by the existence of a strong neuropsychopathic heredity; whilst in the pathological form (the cerebroplegia of Freud and Tanzi) these characteristics are reversed. Pellizzi's studies lead him to dispute the accuracy of the correspondence of these clinical groups with the forms that require to be distinguished upon biological grounds. He adduces numerous facts in support of his contentions, such as that tuberous sclerosis, though without doubt of developmental origin, is accompanied by epilepsy; that there are observations which go to show that spastic cerebral diplegia may be dependent upon a true arrest of development; and that a neuropsychopathic heredity is as a rule present in cases of cerebroplegia. As the anatomical basis of idiocy and imbecility is more fully investigated, the pathological group tends to enlarge at the expense of the developmental. If we could trace back far enough, probably many conditions that we still regard as developmental defects would be found to have a pathological cause. The difficulty of the subject is increased by the fact that an organism defectively developed is much more subject to disease than one normally developed. It is indeed not improbable that deformity and disease are associated in the majority of cases.

The third paper consists of anatomical and histological notes

upon a case of true microcephaly and one of congenital internal hydrocephalus. The histological examination of the brain of the former enables the author to confirm the conclusion of other observers, that in microcephaly there is a scarcity of the medium and large pyramidal cells, and an undue prevalence of fusiform elements, and also, in less degree, of polymorphous and rounded nerve cells. There also seemed to be a superabundance of giant cells. He considers that these histological anomalies are to be regarded as representative of the insufficiency of evolutive energy on the part of the nervous elements of the cortex.

Microscopical examination of the attenuated cortex from the cases of congenital hydrocephalus revealed great diminution in the number of nerve fibres, indistinctness of the cortical strata and paucity of the nerve cells.

The last paper contains a description of the anatomical features in six cases of anencephaly, in some instances accompanied by amyelia. The author concludes that these conditions are dependent upon a primary defect of development of the nervous system which, however, does not manifest itself until after the formation of the secondary cerebral vesicles. The fact that the retina is completely developed, even in its nerve cell elements, proves that the defect of development was not initiated until this period. In two cases of amyelia, and one of hemi-myelia, he found anterior nerve roots with well-developed nerve fibres. Although he admits that this observation may be regarded as supporting the recent view of the development of the nerve fibres from rows of cells, he considers that it is not inconsistent with the teaching of His, as the nerve cells, which gave origin to the fibres, may merely have disappeared. Severe inflammatory alterations were always present in the meninges, but whether the anatomical defects were dependent upon a pathological process in the strict sense of the term, could not be determined.

The same thoroughness, originality, and scientific spirit which marked the first series of the author's studies upon the pathology of idiocy are manifested throughout this later volume. There can be little doubt that the two together form by far the most important individual contribution that has yet been made to our knowledge of the subject.

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# **Review**

of

## **Neurology and Psychiatry**

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### **Original Articles**

#### **THE PRODROMATA OF THE PSYCHOSES, AND THEIR MEANING.**

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IT is a fact in Psychiatry which has by no means been sufficiently recognised, that attacks of mental disease have early symptoms that are often not mental in character ; and it is equally true that in Neurology the fact has not attracted sufficient attention, that all sorts of sensory, vaso-motor and motor symptoms may be the mere preludes to an attack of insanity and not of themselves the real disease. Such prodromata of so serious an event as an attack of insanity are exceedingly well worthy of careful study for many reasons, most notably because the recognition of their true character and their explanation might enable us in many cases to anticipate and possibly to ward off the mental attack. The neurologist who is called in to see a woman suffering from an unusual form of headache, with anorexia, insomnia and obscure paræsthetic sensations, often misses the real point of the case because he does not realise that such symptoms are, in this particular patient, higher-cortical in origin, and may mean an attack of acute mania in a week if nothing can be done to arrest their course. The psychiatrist often considers a mental attack as being sudden in origin, and puts it down as an un-led-up-to mental explosion, when in reality there had been sensory and motor, sleep and other signs which would have proved the

existence of previous auto-intoxication, nerve exhaustion, or other disturbance.

The great fact of solidarity in brain action is one just as much to be reckoned with in psychiatry by the clinician and the practitioner as the more striking and evident facts of localisation and specialisation. The occurrence of a widespread disturbance of action in the highest cortical convolutions, such as takes place in an attack of acute insanity, must, after it has set in, almost of necessity affect the sensory and motor as well as the nutritive and vaso-motor functions of the organ. As a matter of fact, such non-mental accessory disturbances and "bodily symptoms" are nearly always present. But why the oncoming of a nerve storm which is to be in its main course essentially mental should be commonly preceded by sensory, motor and nutritive lesser storms, is more difficult to understand. One may be better able to arrive at the explanation after having looked at the clinical facts. Such combined psychiatric and neurological facts seem specially suitable for discussion in a journal which covers the whole field of brain disturbance, mental and bodily, and which is read by both the nervous and mental specialist and the general physician. It is very certain that it will also require the accurate observations of the family doctor, who has been first called in, as well as the facts seen by the specialist, to settle many of the questions to be discussed in this paper.

*Sensory Symptoms.*—Speaking generally, and including their every degree, disturbances of common sensibility are by far the most frequent and immeasurably the least serious form of neurosis to which nervous humanity is subject. Many women are "seldom without" a headache during some part of every twenty-four hours. In a still greater number of women headaches are easily set up at any time by very slight causes. Indigestion, want of sleep, changes of air and of temperature, worry, harassing domestic incidents, menstruation, over-fatigue, "excitement" of any kind, and alcohol are some of the causes which will inevitably produce headache—slight or severe, bearable or "paralysing"—in many women. Such nerve storms are commonly transitory and not of grave significance. There are many women, too, not otherwise neurotic, who are subject to headaches and other forms of the slighter sensory neuroses. If we could ascertain the etiology and the true meaning of such minor nerve disturbances

we might find ourselves well on the road to the explanation not only of the sensory prodromata of melancholia, but of some of the forms of melancholia itself. Take the following case now under my care. A lady of 50, who has been subject at times to headache and a "weary," painful feeling in the back of her neck and down the upper part of her spine all her life when tired, but was otherwise strong, well-nourished and vigorous, had a special anxiety and much exhausting nursing. Those head and spine symptoms became not only greatly aggravated, but continuous instead of being intermittent. They unfitted her for any exertion, mental or bodily, and in fact she had to keep in bed all day. There was no temperature and no local tenderness. Their persistence still further exhausted her strength, and the next symptom was insomnia. This of course took her down still further, and in a few weeks she became depressed mentally. When the mental pain came on all the bodily pain went off. In the course of three months she was acutely melancholic, and had to be placed in a mental hospital for treatment. She recovered, but the first symptoms of the psychosis passing off was the recurrence of the head and bodily pains. She has had several such attacks since, each one with the same sequence of symptoms. However well she may feel in the intervals, either over-exertion or worry, or a bad catarrhal attack or bronchitis, to which she is subject, will at once bring on the peculiar pains in the back of her head and spine. She then always "has a dread" of her nervousness becoming mental in character. She has the impending feeling of danger and dread which so many melancholics have before an attack. It is essential to know that there is a certain amount of mental heredity in her case. This case is a type so commonly met with as to cover very nearly half the field of melancholia. In most cases the headaches have not quite the character of the ordinary "woman's headache": they are more constant, more intense, more distracting and disabling; the patients will not always admit they are "headaches," but describe them as "peculiar" sensations. Sometimes there are feelings of weight, sometimes of lightness, sometimes a bursting or congested feeling. They are seldom localised, they seldom have the shooting neuralgic character, they are not always relieved by rest and the recumbent position, but rather by fresh air and mild exertion. They have not the character of megrim

and seldom are attended by sickness or vomiting. Their return in the period of convalescence is so frequent as to make me often glad to hear my melancholic patients tell me they are suffering from headache, and I surprise them by saying, "I am glad to hear it, be thankful, you are soon going to get better of your depression." They are most frequently and most characteristically seen as prodromata of the melancholia which follows influenza. Often they assume paræsthetic forms, giddiness, creeping feelings and sensations of weight or lightness, of heat and cold; they are often so peculiar in character that the patients cannot describe them. They complain of "queerness in the head," "soreness," "discomfort," as if they had "no feeling in the head," there is scarcely any paræsthesia that I have not met with. Now, what is the cause of such headaches? What is their precise relationship to the mental disease which succeeds them? Are they toxæmic in character? And if so, what is the source of the toxin? Even if they are toxæmic, this may be merely a secondary and an intermediate stage and not the real primary cause. Through what series of influences does the toxæmia originate? It seems clear to me that we must look beyond the toxæmia, even if this exists, for the real cause of those neuroses of sensibility in most cases. It is quite certain that Dr Haig's uric acid theory does not explain them. Vaso-motor disturbances used to hold the field as explanations of headaches generally. I cannot see in any vaso-motor disturbance a sufficient explanation of the facts: there are no flushings or pallors; the general circulation, though frequently poor, is not uniformly affected. No doubt in fully developed attacks of melancholia, especially those of the worst type, the vascular tension is found to be increased; but taking into account the whole clinical history of such cases, it seems to me one must look to a failure of nutritive and dynamic energising of the higher cortical cells for the real cause. It must be assumed that each neuron, whatever its particular function may be, has an innate power of building up its stores of potential nerve energy (anabolism) and of liberating these stores in a dynamic form (katabolism), these two powers being balanced so that the neuron shall neither become, as it were, too full or too empty. These powers in the sensory neurons are influenced and probably regulated to a great extent from without by muscle action, by the innumerable and constant afferent

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impressions from the skin and viscera, by direct "interference" through other neurons, and finally by the blood current. Any disturbance of this most complicated series of conditions of right working in a "neurotic" subject may no doubt cause the danger signal of pain to be put out. To be "neurotic" or "unstable" in brain working means unrestiveness to the influences from within or without that are adverse to life and health. A cold day "braces" one man and brings on bronchitis in another. A difficulty in life, a calamity even, raises up in one man or woman a spirit of dogged fight to overcome or endure it, in another it breaks down the fighting spirit and sets up melancholia. Now, for the development of the mental cortex and its immeasurable and as yet most mysterious attributes, the constant stimuli on the sensory organs acting first on the great sensory "centres" in the brain and then being transmitted in proper form to the receptive mental centres are absolutely necessary. Mind, in fact, arises through sensations at first. "No sensation, no intelligence." An individual without sensation would necessarily be an idiot. For the great mental functions to work healthily, a healthy sensory system is unquestionably needed. If the sensory system is hereditarily weak or is exhausted or starved or poisoned, then the mental centres that it keeps going must soon exhibit disturbance. Hence I think we have the common sequence of sensory neuroses and melancholia. It is a physiological and psychological fact far too often forgotten, that for a healthy working mind we must have a normal working sensory apparatus. Why does sunshine produce cheerfulness? Or fresh air a feeling of organic comfort? Or the sweet influences of nature mental calmness? The first step in the process is the reception of the sensory impression, the second is the mental effect. The mental areas are only got at through the sensory. The one mostly exists for the sake of the other. The mental areas are like the yolk of an egg, that float in and are entirely isolated by another kind of medium; or rather are like a busy city on an island, its food, its commerce, its electric wires and every source of its life and activity being carried to it through the surrounding sea. A hurricane in that sea comes first; starvation, misery and inactivity in the island come next, as necessary effects. Sensory disturbances arise first, disturbing impressions are sent to the mental cortex, and melancholia or mania, stupor

or mental dissolution come next, as natural physiological sequences. The sensory areas are the least important, the more shallow, and are the gateways to mind, and so suffer first.

By keeping the necessary relationship between the sensory and mental activities of the brain in mind, many of the clinical facts of an attack of melancholia can be explained and many suggestions can thus be obtained for its proper treatment. It must be kept in mind that melancholia in most of its forms is by far the least serious disturbance of the mental areas. It is next mental health, and in the more severe cortical explosions of mania more or less of mental depression comes first of all, thus preceding the deeper mental dissolutions.

The sensory symptoms of pain or of paræsthesia are not confined to the head, though most commonly felt there. I have known many patients to have spinal pains and visceral pains preceding attacks of insanity. I have now a patient passing through adolescent insanity, with periodic exacerbations of mania. For four or five days preceding each maniacal attack, she regularly has severe toothache. The sensory prodromata of mental disease may take the form of disturbance of special sense functions. I had lately a lady who had marked aggravations of eyesight, symptoms from which in minor degrees she had always suffered before she became melancholic. Nothing is more common than a supersensitiveness to light and noises. I have had cases where *tinnitus aurium* and buzzing sounds in the ears preceded attacks of mania. I had lately a woman who became almost deaf for months before she became melancholic. I have seen hallucinations of vision and hearing develop for weeks before the other mental symptoms came on, the patients knowing then that they were hallucinations. Flashings of light before the eyes are very common prodromata to attacks of mania. I have known the sense of taste changed, so that a man could not taste his food or his wine before he became maniacal.

*Motor Disturbances.*—Many mental cases have symptoms which show that the motor centres are disturbed before the mental areas are involved. I have known attacks of acute insanity preceded by general convulsions; but this is rare, though localised twitchings are common enough. That general condition of motor instability known as "fidgets" and muscular unsettledness is exceedingly common. I have met with a marked

alteration in the handwriting. I have also seen a patient quite amnesic for days before the mania came on. The most marked and characteristic motor prodromata of insanity consist of changes in the facial and eye expressions. The muscles which give those expressions are very small, but most highly innervated, and having keen reactivity and wonderful power of co-ordination with each other far beyond any other muscles in the body. Being the mind muscles *par excellence*, they represent emotion and mental action with amazing subtilty and force. Their very subtilty and complications have as yet defied a scientific nomenclature to describe them. Before an attack of mental disease, they often become changed in activity in all sorts of ways. The most common effect is a slowing of their mental reflexes and a deadening of their subtle mechanism. The man about to become insane is commonly changed in facial and eye expressions before his "mind gives way." His eye is either dull or listless, or the cornea has the feverish light on it that accompanies acute insanity. The brow is furrowed, or fixed "lines of care" show themselves on the face of the patient who is approaching melancholia; the play of feature that gives beauty and interest to many faces is no longer seen. There is often an "abstracted" look in the face, which really means that the muscles of expression are morbidly at rest. In this state feeling is purely subjective, with few objective facial or eye accompaniments.

*Neurasthenia*.—That vague, but real enough, condition, which Beard called "neurasthenia," frequently precedes mental attacks. It is, as we all know, difficult to describe or classify, but it means nervous and nutritive exhaustion and unreactiveness to usual physiological stimuli. It certainly has a close kinship to the psychoses in its nature.

*Insomnia*.—There is no symptom more common as a prelude to the acute insanities in almost every form than insomnia. It often precedes the mental attack by many months. What is its cause? What is its pathogenetic explanation? What is its significance? These are questions that have been asked by psychologists and by general physicians from the earliest times, but no satisfactory answers have yet been given. The mystery that has shrouded the physiology and psychology of sleep has never really been lifted by any of the theories which have been put forward for its explanation. But one thing seems quite

certain, the mechanism of periodic brain rest in sleep is most intimately related to the mechanism of mental activity in the brain cortex. The whole body sleeps more or less, but the cortical vehicles of mind have their proper functions more completely suspended than any other organs during sleep. In no class of diseases is the sleep function so disturbed and so much an essential part of the disease, and so constant a prelude, as in the psychoses. When the mental functions are about to be disturbed, sleep is disturbed, may be put down as an axiom. All sorts of vascular theories have been put forward to explain sleep and sleeplessness; I am absolutely convinced that these do not explain the matter. We must look to the cortical cells themselves and to their essential physiological qualities for an explanation of sleep and its disturbances. When the proper balance between anabolism and katabolism is disturbed in the mental area, then you have insomnia, together with mental disturbances, nightmare, disturbed and distressed dreaming, and night terrors, all those being common preludes to mental attacks. Many interesting changes in the kind of sleep and in the modes of going to sleep and of waking are met with: the semi-conscious sleep, the unrefreshing dreamy slumber, the short snatchy sleep are common. Some people are afraid to go to sleep, they "have such dreams."

*Hysterical Attacks.*—It is surprising how common hysterical attacks and a general hysterical state are before the advent of what is commonly recognised as insanity in the female sex. That is what might have been expected, for all the best and most recent writers on Hysteria recognise its kinship to mental disease, and put the mental element in it as being of far more importance than used to be admitted. In fact, one may say that some of the best authorities put down this element as being a quite essential one, and the most important of all.

*Circulatory Disturbances.*—All sorts of circulatory disturbances frequently herald an attack of insanity. Fainting fits, weak heart's action, palpitation, alterations of rhythm, striking changes in the vascular pressure, feelings of "sinking at the heart," are all common. I have seen in the "quick-pulsed" melancholic the alteration of the heart action for weeks before the mental symptoms came on, but this is the exception, no doubt. The capillary circulation often loses tone, the cold-

resisting and heat-producing apparatus suffers. Vaso-motor disturbances occur; notoriously in the climacteric woman about to become insane, flushings, "heats," sensations of giddiness long precede the mental symptoms.

*Blood Changes and Leucocytosis.*—As yet we know too little of the blood changes in mental disease to speak of them in connection with the commencement of attacks, but Dr Lewis Bruce has made the observation that after a patient has suffered from certain forms of Mania there is a persistent leucocytosis, and that if such a patient relapse there is a marked fall in the leucocytosis, and especially in the polymorpho-nuclear cell percentage prior to the onset of the attack.

*Nutritive and Digestive Disturbances.*—Nutritive and digestive troubles often precede the mental symptoms for a long time. Indigestion, dyspepsia in every form, attacks of vomiting, anorexia, falling off in weight and muscular flabbiness are all common, especially before attacks of melancholia. Constipation and altered bowel contents in directions pointing to imperfect digestion, primary and secondary, are present in over 50 per cent. of the cases as prodromata of various forms of insanity. A melancholia of the digestive tract in the shape of obstinate constipation and distressed feelings in the epigastric region precedes and accompanies half the cases of melancholia. Toxic symptoms arising from the intestinal contents have attracted much attention lately, and all sorts of bowel disinfectants have been used, such as calomel, salol, etc., in some cases with very good effect. The relief experienced through a smart purge is a commonplace in therapeutics in such cases. I believe many attacks of insanity are warded off by this means, just as attacks of epilepsy are often so prevented. The acuter insanities and general paralysis are specially apt to be preceded by marked intestinal or gastric catarrh.

The modern toxic school has pushed the theory of intestinal infection so far as to attribute most of the cases of melancholia, of acute mania and general paralysis, to the toxic effects of adverse bacteria which most commonly originate in the alimentary canal. If Dr Ford Robertson's theory of general paralysis being due to a specific organism commonly originating in the intestinal tract turns out to be correct, we shall in future have to fix our attention on this region far more than we have hitherto done.

Certainly the extraordinary developments of bacterial life in the mucous membrane of the stomach, intestines and bowel, which he has discovered and to which he has so strongly directed our attention in general paralysis and in senile insanity, are most striking, even if they should turn out to be only secondary instead of primary infections. It is one of the finest examples of the fact that we commonly don't find what we don't look for, that for so many years we should have examined post-mortem so many hundreds of cases of general paralysis and never seen the evidences of this catarrh till Dr Robertson directed our attention to them, and now we seldom come on a case where they do not exist. I have known six stone in weight lost in the six months preceding an attack of insanity. Nothing is a more common advice which I give to my recovered patients, than to weigh themselves every month and go to the doctor whenever they find they are steadily losing weight. I am satisfied from my experience that many attacks of mental disease could be avoided if those digestive and nutritive prodromata were attended to and counteracted. It is not only true, "make a melancholy man fat, as Rhasis saith, and thou has finished the cure," but "keep fat" if thou art prone to melancholy.

*Menstrual Derangements* are exceedingly common prodromata of attacks of insanity in women. Amenorrhœa is particularly common.

*Mental Prodromata.*—Long before such mental symptoms appear as to constitute "mental disease," we often see subtle mental changes, such as changed emotional states, "deadness" of feeling, hyperæsthetic emotional states, morbid anxieties, accentuations of natural temperament, painfully conscious "nervousness," irritabilities, inability to fix the attention continuously on work, loss of energy, stubbornness, antipathies, mental automatisms, morbid suspiciousness, and the beginning of delusions of the nature of which the patient is then conscious. But those may fairly be said to be the psychoses in a minor degree and not really prodromata, but rather integral parts of the attack, though coming before the main symptoms.

I have not gone into the special prodromata of general paralysis. They are too numerous and characteristic to be dealt with in so sketchy a paper as this. They need further study in the light of modern neurological and bacteriological investigations.

*General Considerations.*—A general consideration of the character and frequency of such facts from the physiological and pathological, as well as from the clinical points of view, leads to the inevitable conclusion that an attack of mental disease is commonly not a simple or localised phenomenon. They show the solidarity of action of the whole of the brain and of the whole of the nerve centres in the cord and the special ganglia of the organic systems of the body. They seem to point to the fact that the lower parts of the sensory apparatus very often break down before the mental apparatus in the highest regions. They seem to prove the mental cortex to be the centre of the organism, and teleologically its end. In this way they point to a greater resistiveness against disease in the higher centres. They show that it is chiefly in the brains hereditarily predisposed to the psychoses that this natural resistiveness breaks down, for all physicians know that all the symptoms that I have mentioned as common prodromata of insanity occur frequently in non-predisposed persons without any mental attack following. They point strongly to the importance of a more careful study and attention to such preliminary symptoms in predisposed persons. They emphasise the view that the whole class of "mental diseases" should be regarded and treated, not as local disturbances, but as widespread departures from the normal physiological condition of the whole organism.

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### **ATAXIC PARAPLEGIA AND SPASTIC PARAPLEGIA AS SYMPTOMS OF DISSEMINATED SCLEROSIS.**

By BYROM BRAMWELL, M.D., F.R.C.P.E.,  
Physician to the Edinburgh Royal Infirmary.

IN the great majority of cases of disseminated sclerosis, when the disease is fully or well developed, difficulty in walking, due either to loss of power in the legs or to ataxia, or to a combination of these conditions, is present. In the great majority of cases of disseminated sclerosis the deep reflexes are exaggerated, and Babinski's sign is present, either on one or both, but usually on both, sides. In short, in the great majority of cases of well-developed disseminated sclerosis there is either ataxic paraplegia or spastic paraplegia or a combination of these conditions.

Ataxic paraplegia and spastic paraplegia should be regarded as symptoms, not as definite clinical entities. It is only in very rare instances and after the most rigid exclusion that one is justified in supposing that a spastic paraplegia is primary, *i.e.* is the result of primary sclerosis in the crossed pyramidal tracts. The same statement, if we leave out of account the ataxic paraplegia often associated with anæmia and cachectic states and due to acute or subacute combined sclerosis, applies, I think, quite as forcibly to ataxic paraplegia in young and middle-aged adults.

My own experience shows that a spastic paraplegia or an ataxic paraplegia which appears to be primary, when it occurs in young and middle-aged adults, is in many cases merely the first evidence of disseminated sclerosis. If cases of this kind are carefully watched for a series of years through their subsequent course, it will, I think, be found that in many instances they ultimately develop into cases of typical disseminated sclerosis. I have now had the opportunity of observing several cases of this kind. I append the clinical histories of three typical examples.

*CASE I. Spastic paraplegia; subsequent development of symptoms characteristic of disseminated sclerosis. Post-mortem appearances characteristic of disseminated sclerosis.*

A single woman, aged 22, a cook by occupation, was admitted to the Edinburgh Royal Infirmary under my care on 7th January 1897, complaining of difficulty in walking due to weakness and stiffness in the legs, especially in the right leg.

**HISTORY.**—These symptoms had developed nine months previously. The history showed that eighteen months previously (in July 1895), the patient had a fright; since that date she had never been quite well; she had felt nervous and out of sorts, and had suffered from palpitation. In April 1896, after a long walk (five miles), followed by a heavy day's washing, she felt her legs heavy and tired, and noticed that her knees gave way under her. Shortly after this she felt her feet heavy, and her toes began to scrape the ground as she walked. She then began to suffer from pain in the stomach after eating. She consulted a medical man, who treated her for dyspepsia, nervousness and anæmia. He thought that the difficulty in walking and her other nervous symptoms were entirely functional. After a long rest and holiday, she improved for a time, but on returning to work her walking rapidly became worse. She accordingly applied for admission to the Infirmary.

**STATE.**—On examining her I found that she was a healthy-looking, well-nourished, high-coloured girl, not in the slightest degree anæmic. As I have already stated, she complained of difficulty in walking, due to weakness and stiffness and a tired feeling in the legs. The gait was characteristically spastic; in walking, the toes and soles scraped the ground. The legs, especially the right leg, were rigid. The muscles of the legs were firm and well-nourished. All the movements of the right leg were markedly weak;



those of the left leg slightly impaired in force. The knee-jerks were markedly exaggerated; knee-clonus and ankle-clonus were present in the right leg, and were occasionally elicited in the left. The plantar reflex was active, but the direction of the toe movement was not noticed (the Babinski sign had not then been described). The bladder and rectum were unaffected.

The grasping power of the right hand was less than that of the left (dynamometer, right=65, left=90).

The patient complained of coldness of the feet, of occasionally numbness in the toes and finger tips. The power of localising tactile impressions was impaired on the 2nd, 3rd and 4th toes of both feet. There was no other objective sign of sensory defect or loss (either to touch, pain, heat or cold). The muscular sense was normal. The special senses were normal. The pupils were equal and active.

There was no volitional tremor, nystagmus, speech affection, impairment of vision or optic atrophy.

There was no pain in the back, no bone disease or other obvious cause for the spastic paraplegia.

The DIAGNOSIS was spastic paraplegia, possibly due to commencing disseminated sclerosis.

COURSE OF THE CASE.—The patient remained in hospital for two months and was then discharged much *in statu quo*.

After her discharge, she gradually got worse. She was again readmitted to the Edinburgh Royal Infirmary under the care of the late Sir Thomas Grainger Stewart, and was subsequently sent to the Royal Hospital for Incurables, Edinburgh. At this time the symptoms were characteristic of typical disseminated sclerosis.

She died in the year 1901. The body was examined post-mortem by Dr Alexander Bruce, who tells me that the pathological appearances (naked eye and microscopic) were typically those of disseminated sclerosis.

NOTE.—When the case was first seen the condition was spastic paraplegia, with some loss of power in the right hand, and numbness in the toes and finger tips.

CASE II. *Ataxic Paraplegia; subsequent development of symptoms characteristic of disseminated sclerosis. Death seventeen years after the patient was first seen. No post-mortem.*

A single woman, aged 24, consulted me in November 1884 because of difficulty in walking, unsteadiness of gait, numbness and coldness in the hands and feet.

HISTORY.—These symptoms had developed a year previously, soon after suddenly arrested menstruation due to sitting on damp grass. The patient thought the sudden arrest of menstruation was the cause of her nervous symptoms; and, so far as I could determine, this was the only apparent cause. She stated very definitely, in answer to my questions, that she had been perfectly well and had not felt any of the symptoms until after the arrest of menstruation.

STATE.—The patient was a well-nourished, healthy-looking young woman. She was not naturally nervous, nor, she and her friends said, in the least degree hysterical. Her hands and feet were red and cold. She complained of numbness in the hands and feet and of occasional giddiness. Her gait was somewhat unsteady and stiff (spastic and inco-ordinate); the muscularity was good, but the legs were stiff and weak; the knee-jerks were markedly exaggerated; ankle-clonus was present on the left side; there had been from time to time some little difficulty in micturition ("forcing"). I could detect no objective sensory defects.

There was no nystagmus, no volitional tremor, no speech affection, no affection of vision and no optic atrophy.

**SUBSEQUENT PROGRESS OF THE CASE.**—The patient improved for a time, but then got worse; there were several variations of this sort (periods of improvement and relapse). I next saw her nine years later, in the year 1893; she then presented the characteristic symptoms of disseminated sclerosis—marked volitional tremor, marked speech affection, but no nystagmus.

She died in November 1901, the total duration of the disease being eighteen years. For several months before her death she was completely paralysed and bed-ridden, contractures and bed-sores developed, the sphincters were paralysed, her eyesight was affected (optic atrophy), her memory and mental power were considerably impaired.

**NOTE.**—When this case was first seen the condition was ataxic paraplegia, with coldness of the feet, numbness of the feet and hands, redness of the feet and hands, occasional giddiness, and slight occasional difficulty in micturition.

*CASE III. Ataxic paraplegia; subsequent development of symptoms characteristic of disseminated sclerosis. Death nine years after the patient was first seen. No post-mortem.*

A single woman, aged 26, was admitted to the Edinburgh Royal Infirmary on 28th September 1893, complaining of difficulty in walking due to weakness, stiffness and inco-ordination of the legs.

**HISTORY.**—She stated that she had been a healthy girl until a year previously. She had then bathed in the sea during menstruation. On coming out of the water she felt very cold, and could not get warm all day, but the menstruation did not immediately stop. Very soon after this she began to suffer from occasional giddiness, numbness in the legs, heaviness and stiffness of the legs, inability to control the movements of the legs, and slight difficulty in micturition.

**STATE.**—On examination I found that she was a tall, thin, and somewhat pale girl. Her gait was markedly ataxic and somewhat spastic; there was some weakness in the legs, dorsiflexion of the feet being especially weak; the muscles of the legs were somewhat soft and flabby; the knee-jerks were exaggerated, and ankle-clonus was present on both sides; there was some difficulty in micturition. The movements of the legs were markedly ataxic. Rombergism was present. There was no loss of power and no ataxia in the upper extremities. The patient complained of numbness and coldness of the feet, and of some numbness in the right hand, also of a girdle sensation. On objective examination some anæsthesia and analgesia were found to be present in the legs and trunk.

There was no nystagmus, no volitional tremor, no speech affection, no affection of vision, no optic atrophy.

After remaining in hospital for two months she was discharged *in statu quo*.

**SUBSEQUENT PROGRESS OF THE CASE.**—The patient gradually got worse, and the characteristic symptoms of disseminated sclerosis (marked intention tremor in the upper extremities, head and neck, nystagmus, speech affection), and, in the terminal stages of the case, paralysis of the sphincters, contractures and bed-sores developed.

She died in April 1902, nine years after the commencement of the disease.

**NOTE.**—When this patient was first seen the condition was one of ataxic paraplegia, with considerable anæsthesia and analgesia in the legs and trunk, a girdle sensation, numbness in the hand, occasional giddiness, some difficulty in micturition and Rombergism

**NOTE ON THE CROSSED PLANTAR REFLEX.**

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Physician to the Edinburgh Royal Infirmary.

IN some cases of hemiplegia in which the Babinski sign is present on the paralysed side, irritation of the opposite (unparalysed) sole produces flexion of the big toe. I have observed this somewhat remarkable condition in at least four separate cases. In September 1901, I had an opportunity of demonstrating it in a patient who was at that time in my ward in the Edinburgh Royal Infirmary to Professor Osler and Drs M<sup>c</sup>Crae and Cushing.

At first sight it is difficult to explain why in a case in which irritation of the paralysed (say the right) sole produces extension of the right big toe (the Babinski sign), irritation of the non-paralysed (left) sole should produce *flexion* of the paralysed (*right*) big toe.

The explanation which occurs to me is as follows:—In normal adults, the plantar reflex is, so to speak, “set to flexion.” In normal adults, irritation of the sole produces flexion (plantar flexion) of the big toe on the same, and if there should happen to be a crossed plantar reflex, on the opposite side.

In the great majority, but not in all, cases of hemiplegia (I have seen several exceptions, and why these exceptions should occur I do not see, unless one may suppose that the fibres of the pyramidal tract *connected with the plantar reflex mechanism* are unaffected by the lesion), the plantar reflex on the side of the paralysis is “set to extension” (dorsiflexion), while the plantar reflex on the non-paralysed side is “set to flexion.” In other words, in the great majority of cases of hemiplegia, irritation of the paralysed sole produces extension of the big toe (dorsiflexion) on the side of the paralysis; while irritation of the sole on the non-paralysed side produces flexion (plantar flexion) of the big toe on the non-paralysed side.

It would appear, therefore, that a lesion of the pyramidal tract so alters the mechanism of the plantar reflex that instead of being “set to flexion” (plantar flexion) it is “set to extension” (dorsiflexion).

Consequently I conclude that in those cases in which the peculiar crossed reflex which I have described occurs—in which

irritation of the non-paralysed sole produces flexion (plantar flexion) of the big toe on the paralysed side—the reflex impulse must cross from the opposite anterior horn of the spinal cord without forming a connection with the reflex arc (centre for the plantar reflex arc) in the grey matter of the paralysed side (see Figure).

If this is so, it is a point of some physiological interest, for it shows the course which the crossed plantar reflex takes through the spinal cord.

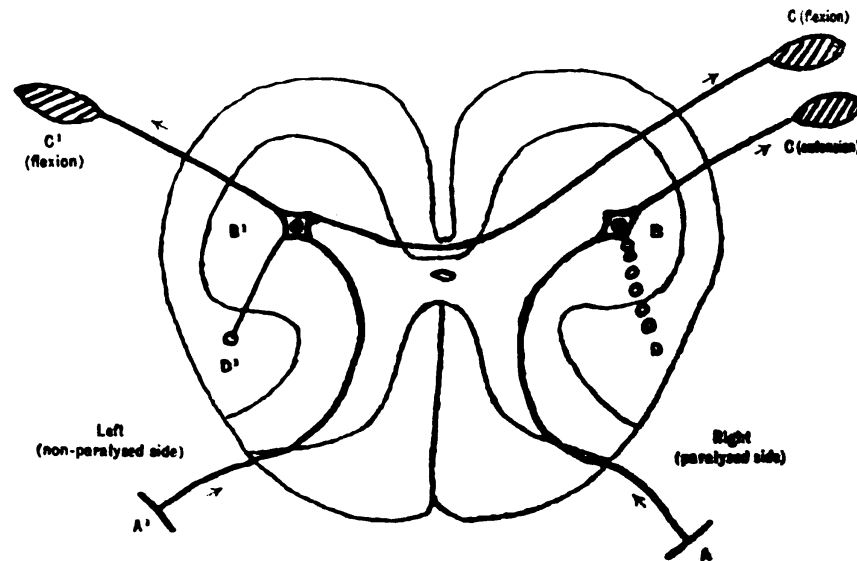


Figure showing the condition of the plantar reflex in cases of hemiplegia and the supposed course of the crossed plantar reflex.

If this theory is correct, one ought to meet with cases of hemiplegia, in which irritation of the paralysed sole produces *extension* of the big toe on the *non-paralysed* side, but in which the non-paralysed side remains "set to (the normal) flexion," i.e. in which irritation of the non-paralysed sole produces plantar flexion of the big toe of the same (non-paralysed) foot. I have not yet met with such a case. If it occurs it will, I think, corroborate and demonstrate the correctness of my theory as to the course in the cord which the crossed plantar reflex takes.

Cases of hemiplegia are occasionally met with where there is a *double* extensor response; such cases are not, of course, available for the experiment—they do not afford any evidence of the correctness of my theory; for in them the plantar reflex on both sides is "set to extension." They correspond to the

well known but not uncommon cases of hemiplegia in which the knee-jerk is exaggerated, and in which there is often ankle-clonus on *both* sides.

The patient is paralysed on the right side, and in consequence of the lesion in the right pyramidal tract (diagrammatically represented by the degenerated fibre which passed from D to B—the reflex centre) the plantar reflex mechanism on the right (paralysed) side is “set to extension.”

On irritating the right (paralysed) sole (A), a reflex impulse passes through a nerve cell in the right anterior horn of the spinal cord down to the right foot (C), and produces extension of the right big toe (the Babinski sign).

On irritating the left (non-paralysed) sole (A<sup>1</sup>), a reflex impulse passes through a nerve cell in the left anterior horn of the spinal cord down to the left foot (C<sup>1</sup>) and (since there is no lesion in the left pyramidal tract, the fibre passing from D<sup>1</sup> to B<sup>1</sup>, in other words since the plantar reflex on the left, non-paralysed, side is in its normal condition—“set to flexion”) it produces flexion of the big toe on the left side.

But when the crossed reflex which I have described is present, irritation of the non-paralysed (left) sole (A<sup>1</sup>) produces flexion of the big toe on the right (paralysed) side (C).

I presume that the fibre which carries this crossed reflex passes from the reflex centre in the left anterior horn of grey matter (B<sup>1</sup>) through the anterior commissure of the spinal cord (or possibly through the grey matter in front of the central canal), direct to a motor nerve root and so on to the motor nerve on the right side, *i.e.* without forming any connection with a nerve cell (reflex centre) on the right side of the spinal cord.

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## Abstracts

### ANATOMY.

**ON THE SIGNIFICATION OF ABNORMAL FIBRE BUNDLES (390) IN THE CENTRAL GREY MATTER OF THE MEDULLA OBLONGATA.** A. PICK, *Névrose*, Vol. v., F. 2, 1903, p. 153.

A CAREFUL examination of the medulla oblongata of an idiot showed the presence of three abnormal bundles of myelinated fibres. These were :—

(1) The set commonly known as "Pick's bundle" lying above the decussation of the pyramids, immediately central to the neck of the posterior horn; these fibres were traced upwards to the level of the *striae acusticae*.

(2) A smaller set of fibres, which at the upper limit of the level of the pyramidal decussation lay in the central grey matter, near the root of the posterior horn; these fibres were traced upwards, and were found to occupy a position in the grey matter at the floor of the fourth ventricle before they became indistinguishable; below, they passed with Pick's bundle into the lateral column of the cord.

(3) Pyramidal fibres which split off from the surface of the medullary pyramid, wind round the olive with the external arcuate fibres, and make their way to a position near the hypoglossal nucleus.

Pick concludes that the second set are fibres descending to the lateral column of the cord from the corresponding posterior longitudinal bundle; whilst the third set are the "fibres pyramidales homolaterales superficielles" described by Madame Déjerine.

The paper is illustrated by many micro-photographs.

STANLEY BARNES.

## PATHOLOGY.

**THE SECONDARY DEGENERATIONS RESULTING FROM A  
(391) CASE OF SOFTENING OF THE CEREBELLUM.** V. NEGEL  
and A. THEOHARI, *Rev. Neurolog.*, Oct. 15, 1903.

THE case reported is that of a man aged fifty years. The clinical aspect is reported in detail, and was typical of a negative lesion of the cerebellum.

The area of softening occupied the whole white centre of the left cerebellar hemisphere lying between the cortex and the dentate nucleus. The upper anterior part of the dentate nucleus was involved. A small spot of recent softening was found also in the right occipital lobe in the region of the cuneus.

No degeneration could be traced into the spinal cord. The only degenerations below the cerebellum being in the arciform fibres going to both olives—chiefly the left, and in both restiform bodies—chiefly the left.

The left superior cerebellar peduncle was much degenerated, and terminated in the red nucleus. The middle cerebellar peduncle was also degenerated.

JAMES COLLIER.

**CONCERNING SOME SUPPOSED TOXIC AND THERAPEUTIC  
(392) PECULIARITIES OF THE BLOOD SERUM OF EPI-  
LEPTICS.** SALA and ROSSI, *Neurolog. Centralbl.*, 1903, p. 852.

THE substance of this paper is preceded by a criticism of Ceni's experiments (*Riv. Sper. di Freniatria*, 1901, f. 344), according to which the latter were conducted without proper precautions, i.e. the patients were under the influence of bromides before and after undergoing the serum treatment, which also did not extend over sufficiently long periods of time. The authors took the following precautions: (1) Withdrawal of all medicinal treatment during a preparatory observation period; (2) Strict regulation of diet and exercise during the preparatory period and also during the period of serum treatment; (3) Record of weight and analysis of food and excretions. The serum was obtained by blood-letting from epileptics who had been for a considerable period without medicinal treatment. The injections of serum were made into the gluteal region. A record is given of five cases of acquired epilepsy, four of the patients varying in age from 18 to 23, while Case 4 was aged 43. Cases 1, 2 and 4 were treated with their own serum, Case 3 with serum derived from Case 4, and another epileptic, Case 5, with the serum of Case 4.

All the observations made are minutely recorded, the authors arriving finally at the following conclusions:—

(1) In no case did the injection of epileptic blood serum exert a beneficial influence upon the course of the disease; (2) No toxic symptom or other reaction was observable; (3) There was no abnormal variation in the general metabolism of the body. The symptoms of these five cases afterwards rapidly yielded to ordinary treatment (bromides, etc.).  
H. LE FANU.

**AN EXPERIMENTAL CONTRIBUTION TO OUR KNOWLEDGE  
(393) OF PHAGOCYTTIC PROCESSES IN THE CEREBRAL SUB-  
STANCE.** U. CERLETTI, *Ann. dell' Istituto Psichiat. della  
R. Università di Roma*, Vol. i., 1901-2.

**UPON NEURONOPHAGIA AND SOME NORMAL AND PATHO-  
(394) LOGICAL RELATIONSHIPS BETWEEN THE NERVOUS  
AND NON-NERVOUS ELEMENTS.** U. CERLETTI, *Ann. dell'  
Istituto Psichiat. della R. Università di Roma*, Vol. ii., 1902-3.

THE earlier of these papers is a preliminary note in which the author records the first results of a series of experimental researches undertaken with the object of ascertaining what cellular elements perform the phagocytic functions within the brain in pathological conditions.

The experiments, which were carried out upon rabbits, con-

sisted in the injection into the cortex of various finely granular substances, of which Chinese ink was found to be the most suitable. The tissues were secured at periods varying from a few hours to fifty days after the injection. The results of these experimental researches lead the author to conclusions which differ considerably from those that have generally been drawn by previous observers. Although within the first few days after the injection black granules could be observed within all of the cellular elements of the cortex, he considers that as regards the nerve cells and neuroglia, the absorption of the pigment is to be attributed merely to an attraction due to nutritive currents, or to an agglutination of the foreign particles in a more or less superficial zone of the protoplasm. On the other hand, a true phagocytic action, associated with amoeboid movements, was clearly manifested in elements which he regards as migrating cells of the vascular connective tissues. These cells were found to be loaded with ink granules within a few hours of the injection having been made, and they continued to exhibit the granules until the reparative process had been completed. Leucocytes did not play any necessary part in the process.

The more recent paper contains a very full account of an elaborate series of investigations designed to test the validity of the grounds for the theory of the occurrence of a process of "neuronophagia." This term, Cerletti explains, properly signifies a mode of phagocytosis by which degenerated nerve cells are attacked, digested and destroyed by cellular elements which accumulate around them. Marinesco, who introduced the term, has asserted that whenever the achromatic substance of the nerve cell suffers injury, there is a stimulation of the surrounding neuroglia cells to phagocytic activity; they hypertrophy, proliferate and become aggressive, invading and devouring the substance of the degenerated nerve cell. The authors who have discussed the question of neuronophagia may be divided into two groups, consisting of those who maintain that the phagocytic cells are of ectodermic origin (neuroglia), and those who hold that they are mesodermic elements (leucocytes or connective tissue cells).

Cerletti regards the distinction of mesoglia cells and neuroglia cells as one that has not yet been satisfactorily established, and adopts the view of Weigert, Degenkolb and Nissl that the vascular tunics are the confines of the mesodermic and ectodermic tissues of the brain. He endeavours to answer two questions which naturally arise in considering the question of the occurrence of neuronophagia: first, Are the rounded nuclei normally to be observed in the vicinity of the nerve cells, neuroglia cells or white-blood corpuscles? and, second, In what pathological conditions can there be an infiltration of the tissues of the central nervous system

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by cellular elements endowed with phagocytic powers? In regard to the first of these questions, he rejects the view, held by many, that the small nuclei to be observed around the nerve cells represent leucocytes, and endorses the opinion of Nissl and others that they are simply neuroglia nuclei. He finds that in perfectly normal conditions these neuroglia nuclei often have the appearance of being partially embedded in the protoplasm of the nerve cell.

His investigations bearing upon the second question have extended over a very wide range of material, including that obtained after the production of various experimental lesions and tissues from cases of tetanus, general paralysis, acute delirium, senile dementia, senility, acute myelitis, etc. He concludes that a neuronophagia in the sense of Marinesco does not occur. He has found no evidence that the peri-cellular neuroglia cells assume any special relationship to the disintegrating nerve cells. Degenerated nerve cells appear to be capable of undergoing complete disintegration apart from any phagocytic action. In some instances in which there are severe lesions affecting the nerve cells, the surrounding neuroglia no doubt undergoes modifications of a progressive nature; but this is not a constant change and it has not the characters of a phagocytic action upon the nerve cells. For the most part, the neuroglia cells hypertrophy and proliferate secondarily; when the nerve cell has already practically disappeared, they tend to fill up the cavity previously occupied by it. He further concludes that leucocytes do not commonly exercise a phagocytic action upon degenerating nerve cells. The authors who have maintained an opposite opinion have, he thinks, simply mistaken neuroglia nuclei for leucocytes; nor can he support the conclusion of Metchnikoff, that the removal of involuting nerve cells is effected by the action of macrophages.

A true phagocytic process does, however, occur when there is locally complete destruction of the nervous tissues. It consists in an invasion by connective tissue elements which assume the form of fat granule or epitheloid cells. It can hardly, however, he thinks, be described accurately as a process of neuronophagia, because it is directed against all the tissue-elements that have undergone disintegration in the necrotic focus, and not merely against the nerve cells.

W. FORD ROBERTSON.

**INCIPIENT TABES. NEUROMA AND HETEROTOPIA OF THE  
(395) SPINAL CORD. A FOCUS OF ACUTE MYELITIS. REBIZZI,  
*Riv. di Patol. nerv. e ment.*, F. 10, 1903, p. 433.**

*History of the Case.*—D. C. contracted syphilis when about 21 years of age; was twice certified insane, and died of the second attack.

When first admitted he was noticed to be suffering from Tabes; gait ataxic, complete abolition of patellar reflexes, pupils non-reactive to light or accommodation, very well marked myosis, Romberg's symptom present. His sight was well preserved, but general sensibility slightly diminished. Just before his death—during a remission of his mental symptoms—whitlows developed, terminating in the loss of some of the finger nails. No other trophic disturbance was noted.

The spinal cord was found to be diminished in thickness, and there was slight thickening and opacity of the meninges, more marked, however, in the posterior region. After several months' immersion in Müller's fluid the cord was examined.

*Lumbar region.*—In the posterior column there was a central area of almost complete degeneration, surrounded by fibres slightly degenerated. The degenerated area involved Trepinski's second and third fibre systems. Posteriorly the degeneration was more marked, for here the fibres of the second system are more numerous. The fibres of the first system lying along the septum and anteriorly were intact.

*Dorsal region.*—The area of degeneration affected the fibres lying along the internal margin of the posterior horn and spread somewhat into the mid-region of Burdach's column, thus involving the second and third system. In Goll's column there was some degeneration, due partly to implication of the latter system.

*Cervical region.*—The fibres of the second and third system were much degenerated.

On passing further up the cord, the author found that at the lower part of  $C_6$  the fibres of the third system lying in Burdach's and Goll's columns were preserved. These could be distinctly followed as far up as  $C_8$ , where, however, other conditions besides the Tabetic process came into play.

The author rejects the various theories that have been advanced as to the causation of Tabes—*i.e.* that the cord lesion is preceded by degeneration of the roots or of the spinal ganglia, or is secondary to meningeal or vascular changes. From the examination of his case, he is inclined to the view that, acting as a predisposing factor in Tabes, there is a special vulnerability of certain fibre systems in the posterior columns, acquired through bad development. This vulnerability or congenital weakness may be caused by intoxications, infections, circulatory disturbances, etc., occurring during embryonal or foetal life; and the case in point supports this view, as there were present distinct developmental irregularities—*e.g.* heterotopia of grey and white matter, small size of cord, and perhaps other developmental alterations in the fasciculi attacked and destroyed by the Tabetic poison. Tabes may thus be described as an affection of weak foetal systems.

Rebizzi admits that the degenerated areas in *Tabes* correspond to a large extent with the embryonal systems of Trepinski, but mentions many factors which add to the degenerative process and prevent one from adopting a classification of Tabetic varieties corresponding with Trepinski's areas — *e.g.* syphilitic or other changes in vessels, changes in meninges, destruction of a nerve cell group leading to degeneration of an endogenous system of fibres.

The changes in the spinal ganglia are described in detail, and the author's opinion is that these are later phenomena, occurring after the onset of the Tabetic process. Should any of the spinal ganglion cells die, then, naturally, degeneration of their central prolongations will add to the cord lesion.

A description of the heterotopia here follows.

Between the end of  $C_3$  and the beginning of  $C_4$  there was asymmetry of the grey matter on one side. The extremity of the anterior horn was much enlarged, and its posterior and external part concave. The posterior horn of the same side was elongated and thinned, except at its extremity. In the external curve of the posterior horn there were many bundles of fibres more deeply coloured than the rest of the cord. Some of the fibres were normal, others altered.

Some sections below this level the posterior horn was almost invisible, and the above described fibres had passed internal to it, until towards the lowest level of  $C_4$  they lay along the internal margin of the posterior horn, and occupied about one-third of the posterior column. At the posterior part of this bundle there was a small pale area, very rich in medullated and non-medullated fibres, neuroglia, and small nerve cells, similar to those usually found in the posterior horns. A little further down the cord the bundle diminished in size, and at the lower end of  $C_5$  it lay again to the outside of the posterior horn, and finally disappeared. It contained two kinds of medullated fibres. One type formed by fibres which ran in wavy lines and formed a network. These were situated especially anteriorly and round the margin of the bundle. They were derived from the nerve cells in the posterior part, were larger than normal fibres, and passed into the lateral columns. A second type of fibres of normal aspect occupied the centre of the bundle and passed also into the lateral columns. These were probably fibres displaced from the crossed pyramidal tracts.

From the upper end of  $C_8$  to the lower end of  $D_2$  acute myelitis was observed occurring in three foci, triangular in shape, with their bases towards the periphery of the cord. In the meninges there were inflammatory changes, and in the areas of myelitis, extravasation of red corpuscles, slight perivascular infiltration,

enlarged and distorted axis cylinders, myelin tumified, nerve cells much altered and many of them destroyed.

*Conclusions.*—1. The author's case supports the view of Trepinski, that Tabetic degeneration may be localised in various foetal systems. This absolute distribution is often altered, however, by primary or secondary degeneration of many scattered fibres, due to death of spinal ganglion cells, or to a direct slow lesion of some of the fibres in the roots.

2. In the cord there is a true neuroma, whose fibres start from a grey nucleus included during foetal life, and which has taken up an abnormal position; there is also an alteration in some of the fibres belonging in all probability to the crossed pyramidal tract.

3. The diminution in size of the cord indicates an arrest in development.

The difference between acute and chronic degeneration of both fibres and sheaths are discussed in detail. DAVID ORR.

**ON THE HISTOGENESIS OF GLIOMA OF THE RETINA.**  
(396) VITTORIO SCAFFIDI, *Virchow's Arch. f. patholog. Anat.*, Bd. 173, 1903.

DR SCAFFIDI gives a detailed description of the histological characters of three of these tumours. The publications of Greeff and Hertel, in which the retinal gliomata were definitely declared to consist of neuroglial and ganglionic cells, have not been fully confirmed by later observers. Still it may be urged that a positive result in the staining of branched neuroglia cells such as that achieved by Greeff is much more convincing than mere failure to demonstrate them; a failure which has, I fancy, been the experience of most of those interested in this branch of pathology.

Dr Scaffidi has been bold enough to publish his failure to demonstrate these cells by any of the methods most in vogue, for example those of Bevan Lewis, Golgi, Weigert, Mallory, Robertson, Yamagwa and Benda.

The first tumour examined was in the initial stage of development. It was obtained absolutely fresh, and under other conditions favourable for successful neuroglial staining. Dr Scaffidi thinks that Greeff's positive results may be explained by accidental staining of normal retinal elements enclosed in the tumour. These have been described as neuroglial and ganglionic cells essential to the tumour formation, while in reality their presence was a mere accident.

In the second tumour, the "rosette" formations, on which Wintersteiner has laid so much stress, were demonstrable, though not quite typical in appearance. Dr Scaffidi does not assign to them any important rôle in the development of the growths. He

is inclined to doubt the presence of a limiting membrane and the existence of remains of rods and cones in these structures.

Dr Scaffidi would derive the retinal gliomata from mesoblastic, not epiblastic elements, basing his theory on the newer views of a double origin of the neuroglia. He would apply the name "mesoglioma" to these growths, as indicating their origin from the mesoglia. Certain cases of *retinitis proliferans* may, he thinks, be more analogous to the gliomata of the central nervous system.

J. V. PATERSON.

### CLINICAL NEUROLOGY.

**FOURTEEN CASES OF SPASTIC SPINAL PARALYSIS OCCUR-  
(397) RING IN ONE FAMILY.** WILLIAM G. SPILLER, *Philadelphia  
Med. Journ.*, June 1902.

CASES of a spinal form of family spastic paralysis are rare, and the author gives a good resumé of the literature of this subject in the first part of his paper.

The author reports fourteen such cases occurring in one family during five generations. Males and females were affected in almost equal proportion, and as a rule the condition manifested itself before or about the fifth year of life, although in one case it commenced as early as the eighteenth month. He describes two cases which he has examined; in both there was simply a spastic paresis of the lower limbs, with some contractures. The deep reflexes were increased with knee and ankle clonus, the plantar reflexes were extensor in type. In none of the cases was there any mental affection. The cranial nerves, upper extremities, sphincters and sensory system were normal in all the cases.

T. GRAINGER STEWART.

**LESIONS OF SYRINGOMYELIA FOUND AT THE AUTOPSY OF  
(398) A GENERAL PARALYTIC.** A. JOFFROY et GOMBAULT,  
*Rev. Neurol.*, 30th Sept. 1903, p. 913.

THE writers record the case of a man, aged 36, with specific history, who showed typical signs, physical and mental, of general paralysis. The knee-jerks were abolished; his movements showed lack of co-ordination, resembling that seen in disseminated sclerosis; there were trophic changes of the skin, especially just before death, but no objective sensory disturbances; the rectal temperature became gradually lower for a week before death, finally reaching 26° C.

At the autopsy, changes characteristic of general paralysis were found in the cerebrum and its meninges, but no gross focal lesion. The inner surface of the spinal dura mater was adherent to the cord in the dorsal region; through the whole length of the cord the outer layer of the pia mater was infiltrated with leucocytes,

and a similar infiltration was noted in the walls of the vessels of the pia mater, especially in the lumbar region, and also in the spinal roots; the nerve fibres of the anterior roots were quite intact, while those of the posterior roots were somewhat less numerous than normal. The antero-lateral region of the cord was intact throughout, with the exception of a small area of degeneration of Gower's tract in the superior cervical region. There was degeneration of the posterior columns, very similar to that seen in recent tabes. The only lesion found in the grey matter of the cord was in the posterior commissure; throughout the dorsal and lumbar regions, but more especially in the lower dorsal region, the central canal was dilated and surrounded by a mass of proliferated neuroglia; the dilated canal was covered by a layer of cylindrical epithelial cells, although at some parts this lining was entirely absent from the wall of the canal.

This case therefore illustrates the development of general paralysis in a syringomyelic subject. The comparative rarity of the association of these two conditions is one reason for publishing the case, but the writers think it has an additional interest and value as bearing on the pathogenesis of general paralysis. It supports the view that the cause or causes (*e.g.* syphilis), which determine the pathological lesions underlying general paralysis, can influence only a nervous system already predisposed; in this particular instance, the predisposition is shown by defective development, viz., the congenital abnormality of the medullary ependyma. It is taken to be, on anatomical grounds, in favour of the opinion held by the writers as well as by Schlesinger, Redlich and others, that general paralysis is "une maladie dégénératrice."

ASHLEY W. MACKINTOSH.

#### **ARTHROPATHY OF THE VERTEBRAL COLUMN IN TABES.**

(399) WILLIAM G. SPILLER, *Am. Med.*, vol. iv., 1903, p. 701.

THE author describes a case of Tabes in a woman aged fifty-nine, in whom besides arthropathies of the left foot, left knee, and right shoulder, there was pronounced skoliosis and lordosis in the thoracico-lumbar region which had come on gradually during the course of four years. There is no further description of the spine. References to several other cases in the literature are given.

H. DOUGLAS SINGER.

**CONCERNING AN ABDOMINAL "SYMPTOM-COMPLEX" IN  
(400) DISEASE OF THE LOWER PART OF THE SPINAL CORD,  
ITS ROOTS AND NERVES.** H. OPPENHEIM, *D. Zeitschr. f.  
Nervenheilk.*, Bd. 24, 1903, S. 325.

In this paper the author describes disturbances of the motor, sensory and reflex functions of the abdominal wall which are met

with in disease of the lower part of the spinal cord, its roots and nerves. The literature of paralysis of the abdominal wall resulting from disease of the lower neurons is reviewed, and the scant attention which the subject has hitherto received is insisted upon. Three cases of abdominal paralysis dependent upon a neuritic or neuromyostic process are then described. In the first case there was bilateral paralysis of the abdominal muscles associated with paralysis of other muscles. The morbid process, which was obviously a neuritis, followed a gastric fever (probably typhoid). In the two other cases there was a unilateral paralysis confined to the muscles of the abdomen, together with local objective sensory disturbance. In both cases the paralysis was evidently due to a unilateral neuritis of the lower dorsal nerves. In neither instance was the etiology very clear. In one case a history of pains and the presence of some tenderness of the nerve trunks of the lower extremities suggested that possibly the unilateral abdominal paralysis might be an abortive form of polyneuritis.

A number of cases of multiple neuritis described by different writers, in which the abdominal muscles were also affected, are referred to. Reference is made to an interesting case recorded by Frederick Taylor in which abdominal herpes was associated with local paralysis of the muscles of the abdomen on the same side.

Oppenheim summarises the symptoms and signs of unilateral abdominal neuritis. The following is a brief resumé of his conclusions. After a febrile process or without obvious cause, pain is felt in the region of the lower intercostal nerves on one side, affecting, it may be, other parts as well. There is tenderness of the corresponding nerve trunks. There may be paræsthesia. Finally, paralysis of the corresponding portion of abdominal muscle develops.

*Objectively.*—The abdominal reflex is lost on the affected side. There is hyperæsthesia or anæsthesia for tactile and painful stimuli in the skin area which corresponds to the affected nerves. The navel deviates to the sound side. The paralysed side of the abdomen appears somewhat swollen, especially during acts which necessitate forced expiration, such as coughing, straining, etc. Finally, R. D. may be demonstrated in the affected muscle. If the affection is bilateral, coughing, sneezing and other forcible expiratory acts are interfered with, and the patient is unable to raise himself without his hands from the supine to the sitting position.

The author then relates a number of cases of abdominal paralysis in which the paralysis was due either to a spinal nuclear or nerve root lesion, and not to an affection of the upper neurons.

Three cases are especially referred to in order to show that in tumour of the spinal cord pain in the distribution of a nerve root may precede other symptoms by months, or even years. Another

very early sign in these cases was loss of the abdominal reflex on the same side. Unilateral paralysis of the abdominal muscles may appear in cases of spinal tumour before there is any sign of pressure on the cord. Deviation of the umbilicus may occur as a result of loss of muscle tone before there is any actual paralysis, and this loss of tone may account for the absence of the abdominal reflex; on the other hand, where there is actual paralysis, especially where it is diplegic, there may be no alteration in the position of the umbilicus. In two cases of spinal tumour, the first sign of pressure on the cord was thermhypæsthesia in the opposite leg.

Remarking on the segmental localisation of the abdominal muscles, Oppenheim says that if cases of progressive muscular dystrophy are excluded, we possess no observations which prove that a limited lesion of the grey matter, or of the anterior roots of the lower part of the spinal cord, leads to an atrophy confined to a segment of the rectus or oblique muscles. On one occasion Oppenheim had an opportunity of stimulating the eighth dorsal root, and he found that no single segment of the abdominal muscle was caused to contract thereby, but that a contraction of the whole muscle resulted.

In a case of unilateral abdominal paralysis during acts of forced expiration, the umbilicus may move far to the sound side (in one case 10 cm.). In the normal person, Oppenheim finds that there is sometimes some asymmetry, the umbilicus moving from .5 to 1 cm. to one or other side during movements of forced expiration such as coughing.

The electrical examination of the abdominal muscles is often very difficult where there is much adiposity. Schöppenberg has shown that slight differences in the electrical excitability of the two sides of the abdomen may be present in health.

In bilateral abdominal paralysis the abdomen is prominent. The author believes that paralysis of the non-striped muscle of the intestine and stomach may occur, and in part, at least, account for the obstinate constipation and meteorism, which are most troublesome symptoms of a lesion in this situation.

Oppenheim refers to the abdominal reflex at considerable length. His observations show that the reflex is absent in 15 to 20 per cent. of healthy people. The activity of the reflex varies considerably in the same individual under different conditions, such as cold, fever, mental excitement, etc., it may be absent at one time, present at another. It is frequently absent in women with lax abdominal walls, but sometimes also in healthy muscular men.

The greatest caution must be taken in any given case before absence of the abdominal reflex is pronounced to be pathological. Where reflex irritability is increased, the author



states that it is particularly well obtained by stimulation of the skin in the region of the *mons veneris* or root of the penis. In the great majority of cases of tabes he has found this reflex increased, and he suggests, as a possible explanation of the divergence between his experience and that of Dinkler, that his cases were chiefly early cases seen in the consulting-room and in out-patient practice, whereas Dinkler's observations were made on hospital cases, in which the disease was probably more advanced.

EDWIN BRAMWELL.

**THE HEMIPLEGIA OF OLD PEOPLE.** FERRAND, *Thèse de Paris*, (401) 1902, pp. 187, 8 plates.

WITHIN the last two or three years attention has been directed, more especially in France, to an interesting pathological condition occurring with surprising frequency in old people who are the subjects of hemiplegia: namely, the presence of *lacunes* or *lacunæ*, minute centres of disintegration which are found most frequently in the great nuclei of grey substance in the cerebrum. Though described in more or less detail by earlier authors, little notice was taken of them till Professor Pierre Marie, at the Congrès de Médecine of 1900, brought the question more prominently before the medical profession. He has since given the results of his observations in an important monograph (*Révue de Médecine*, May 1901).

A *lacune* in this sense is a minute area of softening, varying in size from a pin's head (or smaller) to a pea, or even a small bean. Usually of a greyish tint, it may be coloured exactly like an old hæmorrhagic effusion; in the centre of the space there is always to be found a vessel, perfectly visible to the naked eye, surrounded by a disintegrating and degenerating mass, semiliquid (probably owing to fluid derived from perivascular lymph spaces), of nerve cells and fibres, neuroglia cells, red and white blood corpuscles, as well as other and larger cells, very constantly present, which, on microscopical examination by Marchi's method, are seen to be, in all probability, leucocytes heavily laden with myelin. Microbic forms are conspicuous by their absence. In the earlier stages of the lacune there is no actual perivascular cavity; the surrounding tissue, however, is rarefied and stains very feebly: the neuroglial meshes are wide and the parenchymatous elements are diminished. The central vessel is always profoundly altered; it is much more permeable; the lumen is reduced; the middle coat is sclerosed, and the outer is chronically inflamed and infiltrated by embryonic cells.

Marie was the first to draw attention to these lacunes as being the determining cause of hemiplegia in old subjects. He considered

that 90 per cent. of all hemiplegias occurring in patients over sixty years of age are "lacunar" in origin. In examining the site of the lesion one is struck by the frequency with which the nuclei of grey matter are affected. In only 13 per cent. are the lacunæ found in the white matter. Further, a remarkable preference is shown for the lenticular nucleus. In 183 observations, it was affected no fewer than 66 times, the optic thalamus 35 times, the caudate nucleus 18 times, the internal capsule 25 times (usually by extension, be it noted), the pons 24 times, the centrum ovale 14 times, and the corpus callosum 3 times.

In his thesis, Ferrand has contributed materially to our knowledge of the subject, and has brought forward an amount of clinical and pathological evidence which is very convincing. He gives details of no fewer than 97 cases of hemiplegia in old men, in 88 of which post-mortem examination revealed the presence of one or more lacunes. In 64 cases the lesion was in the lenticular nucleus.

It is somewhat surprising, considering the frequency of the lesion, that its significance has not been fully appreciated before now. Ferrand is strongly of opinion that faulty technique has much to do with this. He recommends the hardening of the brain *in situ* by the injection of formol, *via* the eyes, as soon as possible after death. He has further found that a horizontal section through the hemispheres by a plane which just touches the under surface of the anterior and posterior extremities of the corpus callosum will reveal, in the vast majority of cases, the presence of lacunes.

The *type* of hemiplegia occasioned by this lesion is of interest and importance clinically. The patient is usually the subject of a sudden, though mild, apoplexy. He is dazed, loses the power of his limbs, and falls to the ground. There is not usually any definite loss of consciousness. When examined he is found to be paralysed on one side, but this paralysis is partial—that is to say, often only one limb is affected; it is incomplete—that is to say, certain movements are still possible in the limb which is paralysed; above all, it is transitory. It may last only for a few minutes, at the most a few weeks. This being the case, it may be difficult to satisfy oneself that the patient has had a hemiplegia at all. Ask him to button his coat alternately with the right hand and with the left. Ask him to close alternately the right eye and the left. These little devices very often reveal which was the side affected. An important symptom is the gait of the patient. Bending a little forward he trails his legs along, the sole of the foot never leaving the ground, one foot being advanced in front of the other only a few inches. This gait, characteristic of pseudo-bulbar paralysis (Brissaud), of paralysis agitans also, is still more characteristic of lacunar hemiplegia. It is known as the *démarche à petits pas*.

One other clinical fact ought to be mentioned, viz., the absence of contracture in the paralysed limbs. An interesting chapter is devoted to the pathogeny of the condition. The care with which the technique of examination was carried out is sufficient to show that the lacune is not an artefact. Clinically and anatomically it is not a hæmorrhage in the ordinary sense of the term, though it is often the cause of one. It is not a softening, a local necrobiosis following on atheroma and obliteration of an arteriole, for the vessel of the lacune remains permeable. The evidence is in favour of its being a chronic secondary encephalitis, developed, so to speak, round the vessel, in connection with which—in the absence of microbic irritation—one must look for the primary cause. That this primary cause is of the nature of an arterio-sclerosis there is much in the way of proof: the diminished nourishment of the part—for the arterioles are terminal—accentuates the pathological condition once it is set up. The rôle which the perivascular lymphatics may play in the pathogeny of the lacune is more problematical, nevertheless it is conceivable that they may become chronically inflamed from retained products of decomposition (“destructive vaginalitis” of Marie).

One other point may be mentioned. From an examination of the lesion in his 88 cases, the author is of opinion that the miliary aneurism of the text-books is infinitely less common than is usually supposed. In 17 per cent. the cause of death was cerebral hæmorrhage, in every case lacunes being manifest. In a year and a half's pathological experience at Bicêtre, where cases of cerebral hæmorrhage are numerous, Ferrand found that there was none in which there were not also lacunes. And as a matter of fact, Professor Marie is now teaching that in old subjects cerebral hæmorrhage is not due to the bursting of a miliary aneurism, but to the rupture of the central vessel of a lacune. S. A. KINNIER WILSON.

**THE OCULO-MOTOR NERVES IN ORGANIC HEMIPLEGIA OF (402) ADULTS.** DESCLAUX, *Thèse de Paris*, 1903.

THE author begins by quoting the classic opinion that muscles which normally act together are not affected in a hemiplegia of organic origin. Nevertheless there have appeared within recent years various contributions to the subject which, to say the least, render some modification necessary. Reference is made to the work of Nothnagel on unilateral paresis of the thoracic muscles in hemiplegia; to that of Babinski on the alterations of tension of the skin folds in the platysma; and to that of Sicard on one-sided paralysis of the abdominal muscles. Hence Déjérine (1901) gives it as his opinion that in a hemiplegia the muscles are paralysed proportionately to their normal force.

Reference is further made to a paper by Mirallier on the widening of the palpebral aperture in peripheral facial paralysis, and its diminution in certain cases of hemiplegia of cerebral origin. From this it would appear that sometimes the third nerve may participate in the paresis or paralysis of the affected side.

With a view to verifying this, the author undertook the systematic examination of the eye muscles in eighteen cases of hemiplegia. He used for the purpose a tube of zinc 24 cm. long, 4 cm. in diameter at the end placed next to the eye, and 1 cm. at the other, together with a series of graduated prisms. The patient was seated in front of a black disc, at a distance of 5 or 6 metres; the zinc tube was then placed close to the sound eye, and the patient looked at the disc through it. It was first ascertained that there was no diplopia present, when a prism was placed in front of the other eye, producing diplopia unless the muscles of this eye managed, by accommodation, to overcome the deviation. The strength of the prism was increased gradually till the eye could not overcome it, and permanent diplopia appeared. The number of the prism was noted. By turning the base of the prism in different directions, the superior and inferior, internal and external recti were successively tested.

From six observations on the normal individual it was found that homologous muscles of the two eyes are equal in strength, that the internal and external recti are more powerful than the superior and inferior, and that the internal rectus is much the most powerful of all.

This might be formulated thus:—

$$\text{Rect. int.} > \text{Rect. ext.} > \begin{cases} \text{rect. inf.} \\ \text{rect. sup.} \end{cases}$$

The author found:—

$$\begin{aligned} \text{Rect. int.} = \text{Rect. ext.} &= \begin{cases} \text{rect. inf.} \\ \text{rect. sup.} \end{cases} & 9 \text{ times.} \\ \text{Rect. int.} > \text{Rect. ext.} &\geq \begin{cases} \text{rect. inf.} \\ \text{rect. sup.} \end{cases} & 8 \text{ times.} \\ \text{Rect. int.} < \text{Rect. ext.} &> \begin{cases} \text{rect. inf.} \\ \text{rect. sup.} \end{cases} & 1 \text{ time.} \end{aligned}$$

In all the cases, the strength of each muscle was diminished on both sides, but especially on the hemiplegic side. The order of the muscles—in reference to their power—was always the same on both sides. The alteration as given above was always most marked in the hemiplegias of recent date. In five of these cases there was ptosis, and in two mydriasis. These symptoms disappeared quickly.

The author discusses briefly the theory of the involvement of the eye muscles in a hemiplegia and gives references to recent anatomical research on the cortical representation of bulbar centres (Roux, 1899).

S. A. KINNIER WILSON.

**MYOSIS IN CERTAIN BULBAR FOCAL LESIONS. HEMI-  
(403) PLEGIA OF THE AVELLIS TYPE ASSOCIATED WITH  
THE OCULAR SYMPATHETIC SYNDROME. CESTAN and  
CHENAIS, *Gaz. des Hôp.*, Oct. 29, 1903.**

THE classical symptoms of a lesion involving the cervical sympathetic in any part of its course are myosis, without any loss of the pupillary reflexes, slight ptosis and narrowing of the palpebral fissure, and some degree of enophthalmos.

In 1901, Hoffmann published three cases exhibiting clinically these symptoms along with others certainly of bulbar origin, viz., crossed hemi-anæsthesia (of face and limbs), paralysis or paresis of the palate on the same side as the facial anæsthesia, with or without paralysis of the vocal cord on this same side, and with or without motor troubles in arm and leg.

In 1902, Babinski and Nageotte described three more cases, in which there were on one side, anæsthesia of the face, paralysis of the palate and of the vocal cord, involvement of the cervical sympathetic; on the other, anæsthesia of the arm and leg, loss of motor power in the leg, and an extensor response.

The authors describe a seventh similar case. The interest lies in the association of cervical sympathetic symptoms with other phenomena indubitably of bulbar origin.

The course of the cervical sympathetic fibres from the cilio-spinal centre of Budge by the superior cervical ganglion, Gasserian ganglion, fifth nerve, and ciliary nerves to the eye is, of course, well known. The existence of an upper or bulbar centre has long been postulated, and some physiologists have described a simple path from the medulla down to the fourth cervical segment (cilio-spinal centre). On the other hand, it has been shown experimentally that after section of the fifth nerve between the medulla and the Gasserian ganglion, *i.e.* before it is joined by the cervical sympathetic filaments, excitation of the distal end produces mydriasis. This (and other evidence) would seem to show that there is an *upper* path for irido-dilator fibres from the medulla direct by the fifth nerve to the ophthalmic ganglion. In accordance with this, the cases recorded all show myosis and facial anæsthesia on the same side.

The hemiplegia is perfectly characteristic, the seat of the lesion being in the posterior part of the medulla (*calotte bulbaire*), probably in the neighbourhood of the fifth nucleus.

This localisation explains in addition the other clinical phenomena.

In Babinski's cases there were troubles of equilibrium and lateropulsion of a cerebellar type, pointing to involvement of the

inferior cerebellar peduncles, which are, of course, close to the area above described.

The remarkable association of palate and recurrent laryngeal paralysis on the same side as the bulbar lesion is known as the syndrome of Avellis (*Berlin. Klin.*, Oct. 1891). Evidence in detail is given to show that the palate is innervated by fibres from the "vago-spinal" nucleus, and that the pathology of this syndrome is a lesion in the neighbourhood of this nucleus. Further, it is probable that the cervical sympathetic arises from the intermediolateral tract in the cord, the continuation of which upwards, known as the solitary bundle, supplies fibres to, or is in direct connection with, these mixed nerves of the lower part of the medulla.

The authors thus explain the symptoms in this very definite entity—this "medullary syndrome" which ought to have a place beside the syndrome of Weber, of Millard Gubler, and of Avellis.

S. A. KINNIER WILSON.

**THE MANIFESTATIONS OF TUMOURS OF THE CEREBELLUM**  
(404) H. DURAT, *Rev. Neurolog.*, Oct. 15th, 1903.

THIS communication is a most excellent resumé of the symptoms of cerebellar disease.

The writer describes the physiological action of the cerebellum as follows:—The functions of the cerebellum are both static and dynamic. The first of these is evidenced by the disorders of equilibrium which occur in experimental and pathological lesions. As regards its dynamic function, the cerebellum is a centre of energy and for reinforcement. It may be compared to a great ganglion placed behind the brain and brain stem, as are the dorsal ganglia with reference to the spinal cord. It constantly produces nervous incitations destined to furnish the neuro-muscular apparatus with potential; to maintain its tone and also its vitality. Further, the cerebellum is important in blending and co-ordinating muscular movements, in such subvolitional actions as the maintenance of the upright position, walking, etc. Consequently, as Luciani has shown, lesions of the cerebellum produce: Asthenia (diminution of the energy of muscular contraction); Hypotonus (flaccidity of the muscles); Astasia (defects of static equilibrium); Titubation; Asynergy; Cerebellar Ataxy.

According to Thomas, the cerebellum is an annex upon the sensory paths which receive both excitations from the periphery and impressions from the higher centres. Under the influence of such diverse excitations, it is the seat of a reaction which expresses itself in the maintenance of equilibrium in the several attitudes and in movements both voluntary, automatic and reflex. In other words, it is the reflex centre for equilibration. That the

functions of the cerebellum are much influenced by sensory impression is proved by the fact that experimental lesions of the cerebellar hemispheres and vermis are followed by more severe and more persistent ataxy if the eyes or the semi-circular canals are extirpated at the same time.

The indication that a neoplasm is situated in the cerebellum are as follows:—

1. Headache, vomiting, vertigo and amaurosis as a rule appear early and soon become intense. A very slow development of symptoms, however, is not rare in cases of cerebellar tumour.

2. Rigidity of the neck, opisthotonus, forced attitudes of the head, and especially an inability to bend the head forwards are most important symptoms.

3. Muscular asthenia and loss of muscular tone are very constant manifestations.

4. Troubles of equilibrium, such as stumbling, a reeling gait and falling.

5. Defects of co-ordination are constant in the upper extremity. In the early stages of the disease they are expressed in a certain awkwardness of movement which may easily be overlooked. In later stages the inco-ordination of volitional movements may be so great as to resemble the intention tremor of disseminate sclerosis.

#### 6. Cerebellar Asynergy.

This condition first described by Babinski is pathognomonic of a lesion of the cerebellum.

It consists in a disturbance of the faculty of associating movements.

For the demonstration of this symptom the following tests are convenient:—

1. In walking the trunk does not follow the movements of the legs. It remains behind, so to speak, and it may be necessary for the observer to draw the trunk forward to preserve the balance.

2. When standing, if the trunk be passively pressed backwards, so as to disturb equilibrium, the thighs do not at once flex to restore equilibrium as in a normal person, but they remain fixed.

3. In the act of changing from the supine to the sitting position, the thighs are markedly flexed and the heels are raised.

4. If when seated the patient makes the attempt to touch with his foot an object placed in front and above him, the thigh and the leg extend at different times, as if the leg were held back and suddenly released.

Again, owing to this asynergy the hand-writing may be seriously interfered with.

Often hemiasynergy exists and indicates a lesion upon the same side.

Where asynergy exists two interesting phenomena are generally to be observed. Firstly, there is a marked increase of static equilibrium in the lower extremities, so that if a patient be told to raise both lower extremities from the bed and hold them still he is able to hold them without oscillation in a state of absolute fixity for a much longer period than can a normal person. The second is an inability to perform rapid successive movements, such as pronation and supination of the forearm. The faculty of executing successive movements rapidly has been termed by Babinski "diadococinesia."

Epileptiform attacks do sometimes occur in uncomplicated cases of cerebellar tumours. Sudden loss of consciousness without the occurrence of convulsion is more common.

Paralysis and contracture do not occur from disease of the cerebellum, but they may arise indirectly as the result of pressure or ventricular distension.

*Reflexes.*—The deep reflexes are increased upon the side of the lesion in a great majority of cases.

Defects of sensibility and of intelligence are not symptoms of uncomplicated cerebellar disease.

*Diagnosis of Tumours of the Middle Lobe and Vermis.*—This rests upon the distribution of the above mentioned symptoms equally upon both sides of the body. Head retraction and opisthotonus are especially prone to occur. The writer points out that tumours confined to the vermis not unfrequently give rise to facial and auditory palsy, as the result of indirect pressure. Sudden death is not of unfrequent occurrence from pressure upon the medulla.

*Tumours of the lateral lobe.*—Upon the same side as the brain is situated the following signs occur: Muscular Asthenia; Cerebellar Ataxy; Hypotonus; Asynergy; Loss of cinetic equilibrium; Increase of static equilibrium; Increase of the deep reflexes.

The head is inclined to the same side, and the face looks somewhat upwards and to the opposite side.

The patient lunges to the same side in walking. He tends to fall to the same side; and if rotation be present, he rotates from the supine position to the prone position over the side of the lesion.

The cranial nerves tend to be affected indirectly in cases of cerebellar tumour in the following order of frequency: the facial and auditory, the sixth, the ninth, the vagus, the spinal accessory and the hypoglossal. The writer states that the affection of these nerves is always upon the side of the lesion.

*Cerebellar Deficit.*—Following Thomas, the writer describes a special syndrome resulting from agenesis and cerebellar sclerosis. It consists essentially of troubles with movements when stand-



ing and walking, whilst there is relative integrity of individual movements of the limbs when the patient is lying supine in a horizontal plane. The patient stands upon a wide base, and oscillatory movements of the head and of the trunk occur. The head and trunk are always inclined either to the right or left, and always to the same side. The lower extremities shake as he stands. In walking the oscillations increase, he advances in a broken line, the body being carried now too much to the right, now too much to the left. He reels and stumbles; but he never throws his legs about as does a truly ataxic patient. The oscillations are often severe enough to cause him to fall, and he becomes fatigued very soon. Speech is usually slow. There is in addition ataxy of the upper limbs and general muscular asthenia and flaccidity.

JAMES COLLIER.

**FURTHER CONTRIBUTIONS TO THE PATHOLOGY OF SENSORY**  
(405) **APHASIA.** A. PICK, *Arch. f. Psychiat.*, Bd. 37, H. 2.

THE patient was German-born, but had been able to also speak Czech perfectly. In September 1898, when he was 72 years old, a cerebral thrombosis occurred. The symptoms were as follows:—General weakness, uncertainty in walking, difficulty in feeding himself, complete loss of memory, complete inability to understand or speak German and great difficulty in understanding or speaking Czech. In a few days he recovered to a certain extent his command over the German language, and then did not materially change until carefully examined in November 1900. He had been deaf for ten years.

He was now found to be very deaf, but could be communicated with by shouting. He was able to understand spoken words a little, could write only very incorrectly; he could not name objects as a rule, nor could he repeat spoken words correctly; his answers to questions were usually irrelevant, but otherwise were not senseless. He was generally feeble, but showed no hemiplegic sign.

The patient died on December 24. It was found that there was softening in the left hemisphere only: it involved the supra-marginal and angular gyri, the hinder two-thirds of the superior temporal and the hinder one-half of the middle temporal convolutions.

The author discusses the case fully and concludes that the peculiar aphasic condition was due to the action of the auditory word centre on the *right* side (that on the left being destroyed) in the presence of a peripheral disturbance of the apparatus of hearing.

STANLEY BARNES.

**THE SENSORY SEGMENTAL AREA OF THE UMBILICUS.**  
(406) WILLIAM G. SPILLER, *Philad. Med. Journ.*, Feb. 8, 1902.

DR SPILLER records a case of fracture of the spine through the upper part of the tenth dorsal vertebra. At the autopsy it was shown that the cord was completely compressed at this level. The ninth dorsal segment and the corresponding nerve roots were not implicated, whereas the tenth dorsal segment of the cord was much softened. Below the level of the fracture the cord was softened and showed inflammatory changes.

During life there had been complete paralysis of the lower limbs, with absence of the knee and Achilles jerks and the Babinski phenomenon. There was, too, complete anæsthesia and analgesia below the level of a line which passed exactly through the umbilicus. The line which separated æsthesia from anæsthesia passed almost horizontally round the trunk, although in the middle line of the back it extended half an inch, and over the right hip one inch above the umbilicus. The dividing line was very sharp; above it sensation for touch and pain was well felt, below it there was complete anæsthesia and analgesia. The author's observation bears out Head's view that the tenth thoracic segment represents the sub-umbilical area, the upper border of which passes through the umbilicus. The observations of Dejerine, Walton, Thorburn, Wickmann and Seiffer are referred to in connection with this point.

The absence of the knee and Achilles jerks and Babinski phenomenon may be explained by the softening of the cord below the level of the fracture. The author remarks that the absence of the Babinski sign in such cases may possibly prove of value in coming to a conclusion as to whether there is disorganisation of the lumbar and sacral regions of the cord.

EDWIN BRAMWELL.

**THE IMPORTANCE OF THE LACHRYMAL REFLEX IN THE**  
(407) **DIAGNOSIS BETWEEN ORGANIC AND HYSTERICAL**  
**ANÆSTHESIA OF THE FACE.** WILLIAM G. SPILLER,  
*Philad. Med. Journ.*, May 17, 1902.

Two cases of hysteria are referred to in which there was anæsthesia of the scleral conjunctiva, but in which irritation of the conjunctiva produced as copious an increase of the lachrymal secretion on the anæsthetic as on the æsthetic side.

A case of anæsthesia of the conjunctiva following removal of the right Gasserian ganglion is mentioned, in which there was no excess of lachrymal secretion on the affected side, produced in consequence of direct irritation. Irritation of the right eye produced very little increase of the lachrymal secretion on either side; irritation of

the conjunctiva on the left side, however, produced great increase in the left eye, little, if any, in the right. The author does not believe that the importance of the lachrymal reflex is fully established by these observations, but he has reported the cases in order to draw attention to the point in the hope that by the observation of many cases the diagnostic value of the lachrymal reflex may be determined. He found that irritation of the nostril was the most satisfactory method of testing the reflex. Dr Spiller points out that Pitres and Gilles de la Tourette have previously noted the preservation of the lachrymal reflex in hysterical anæsthesia of the conjunctiva, but so far as he knows no diagnostic importance has been previously attributed to the phenomenon.

EDWIN BRAMWELL.

(408) **CLINICAL STUDIES.** A. PICK, *Brain*, Summer 1903, p. 242.

I. On "*Dreamy Mental States*" as a Permanent Condition in *Epileptics*.

THIS paper, which is dedicated by its author as a tribute to the "Master of Neurology, Hughlings Jackson," contains a description of two patients in whom epileptic convulsions had been replaced by dreamy mental states, at first temporary, but later becoming almost permanent.

Case 1, a lady aged thirty, had suffered from epilepsy since the age of twelve, the fits having been occasionally followed by uncontrollable impulses. Under treatment the convulsions became fewer and were replaced by attacks of *petit mal* and the peculiar psychopathic condition of dreamy mental states. This latter condition gradually became more and more permanent. The state of mind is well illustrated by extracts from the private diary of the patient, of which the following is an example: "I can trace such a terrible, oppressive, disagreeable confusion in my head during sleep throughout all jumbled dreams. . . . Such a confusion, such pell-mell. Something oppressive, so melancholy. I also have it before going to sleep, on waking, this terrible not to be described, that robs me entirely of clearness." Associated with this mental state there was loss of the sense of smell, and the patient complained of various paræsthesias of the left half of the body.

Case 2 had suffered from typical epileptic convulsions, but had been nearly free from them for many years, during which there had developed a mental condition, almost permanent, very similar to that of the last case, with dreamy conditions, hebetude, decrease of consciousness, terrifying thoughts and loss of personal sensibility, although he asserted that he could not well say what was really happening to him. This patient also kept a diary of his mental condition, of which extracts are given.

II. *On the Pathologically Protracted Duration of Impressions on the Senses as a cause of various disturbances of the Sensory Perception and especially of the Sight.*

Under this heading, Professor Pick attempts to explain certain disorders of the special senses so common among neurasthenics and in the insane. The explanation here given was suggested by the entirely spontaneous remarks of an intelligent lad of fourteen years of age, suffering from traumatic neurasthenia, with regard to his sight. Although to ordinary methods of examination the eyes were normal, both functionally and anatomically, he complained that objects looked peculiar, and added further, when somebody passes him or a vehicle drives past he sometimes *still sees a shimmer afterwards*. This suggestive remark was later supplemented by stating that he had *a sensation as if the object in question were broader than normal*. The author suggests that the variation in the appearance of fixed objects might be similarly produced by the persistence of after-images, because in looking at a stationary object the eyes are usually continuously on the move. Arguing from analogy, variations in the perception of sounds might be produced by increased after-sounds.

III. *On Reduplicative Paramnesia.*

The author here describes a case of a curious type of paramnesia which he proposes to differentiate under the above name, and refers at the same time to a similar case published by himself at an earlier date. The present patient was a woman of sixty-seven years who had been normal until shortly before she came under observation, when she began to lose strength rapidly and developed the condition of presbyophrenia with depressed moods, forgetfulness, fleeting delusions, and illusions of memory. She also had a "fainting fit" which was probably an attack of apoplexy. The paramnesia arose after she had been for three months in the hospital, and consisted in the belief that she had been in two different clinics of similar arrangement and containing many of the same patients. At first she believed the two clinics were in widely different localities, but later said that one was on the floor above the other. She did not, however, duplicate the professor, his assistants, or the attendants.

H. DOUGLAS SINGER.

PSYCHIATRY.

HALLUCINATIONS AND SENSORY MENTAL DISTURBANCES.

(409) MONDIO, *Riv. Speriment di Freniatria*, Vol. xxix., F. 1 and 2, p. 240.

THE study of hallucinations has attracted much attention, and many hypotheses have been offered to explain their character, their seat, their mechanism, and their pathogenesis.

The result of recent work has been to prove the identity of illusions and hallucinations, and the affinity between hallucinations and mnemonic images.

Tamburini, in 1880, affirmed that hallucinations are the result of an irritative condition of the psycho-sensory centres of the cerebral cortex, analogous to that which in the psycho-motor centres produces epilepsy.

The convulsions in the sensory centres awaken the images in simple or complex forms according to the intensity of the stimulus.

Whatever may be the origin of the morbid action, the seat is always that part of the cortex which normally perceives the actual sensations, and which, when excited, can reproduce them in a mnemonic form.

Tanzi, however, recognises two centres, one for the perception of things, and the other for the representation of them. He suggests that an idea formed in the representative centre, instead of ascending and associating itself with other ideas, and projecting itself externally as a movement, flows back to the sensory centres. The relation between the sensory and representative centres is thus reversed.

Others have suggested that hallucinations are due to some abnormal stimulation of a peripheral nerve or its cell.

Without going further into these hypotheses, it may be stated that hallucinations now take a much more prominent part in our conception of mental pathology; in fact, a form of mental disease is recognised, in which the sensory disturbances are the primary element, to which other mental symptoms may become attached later.

The author then describes five cases in which the mental disturbance was preceded by hallucinations: in cases 1, 4 and 5 the hallucinations were both auditory and visual; in case 2 they were auditory alone; and in case 3, visual alone.

Moreover, in case 1 there was simply a hallucinatory condition, which recovered and repeated itself several times; in case 2 a melancholic condition developed after some time; in case 3 a condition of confusional insanity; in case 4 a mild delirium of persecution; and in case 5 a delirium similar to that seen in paranoiacs.

One of the most noticeable features of these cases is that there is little change of the psychic personality, at least for a very long time.

Bianchi describes three groups of cases. The first group includes those which depend on a false perception. In these cases one has to deal with neuropathic brains, vulnerable because of heredity or excesses, whose psychic functions are disorganised by small sensory disorders.

The second group embraces those cases in which vivid hallucinations attack a psychic personality, which, up to the time of the attack, had acted well, and produce a psychopathic state of confusion or melancholy, or a delirium similar to that seen in paranoiacs. But in these last the hallucinations are not primary, and there is not the chance of recovery that there is in the sensory cases.

In the third group he places those cases of stronger mental constitution, in which the hallucinations are varied but not vivid, and in which there is no sign of disturbance of the mental personality for a long time.

With regard to the relation between confusion and hallucination, the author suggests that many of the cases which are now described as examples of confusional insanity, would, if they were seen in an earlier stage, be recognised as secondary to sensory disturbances.

Moreover, if we admit the functional difference of the different provinces of the cerebral cortex, and the different importance which each of these provinces has with respect to the others, corresponding with the different period of their development considered phylogenetically and ontogenetically, it may be possible to group mental diseases according to the province from which starts the first sign of disturbance of the psychic equilibrium. It is interesting to note also that in these cases the hallucinatory states were agitated or apathetic according to the hallucinations present. When there was a simple auditory or visual hallucination alone, the patient was usually apathetic; but when there were multiple hallucinations the patients were in a condition of fright, and showed persistent psychic and motor agitation.

Now, apart from the hypotheses of Tamburini and Tanzi, there can be no doubt that, given a vivid hallucination, the degree of disturbance of the psychic personality will depend on the grade of weakness, hereditary or acquired, of the nervous elements of the brain.

The author then, in the light of his five cases, discusses the theory which Tanzi has put forward to explain hallucinations. According to Tanzi the disorder begins in the psychic zone, and affects the sensory zones secondarily.

This may be sufficient to explain the conditions in paranoiacs in whom the hallucinations are secondary to the psychic disturbances, but it is insufficient to explain these sensory cases in which the disturbances of the sensory zones are primary.

Finally, the author adopts the hypotheses of Tamburini.

R. G. Rows.



**THE MENTAL CONDITION OF PATIENTS SUFFERING FROM**

(410) **TIC.** PELI, *Riv. Speriment di Freniatria*, Vol. xxix., F. 1 and 2.

THE author refers first to a communication made by Meige and Feindel to the Congress at Limoges in 1901.

Meige and Feindel mentioned two characteristics which were always present in cases of tic: (1) A motor disturbance; and (2) a peculiar mental condition with which the motor troubles were closely correlated.

The character of these patients exhibits a variability, a weakness of will, an instability, which are evidenced by sudden excesses and desires, by imperious caprices, by impatience and irritability. Their power of attention is weak, they are emotional, and their affective nature is altered. Another noticeable point is the rapidity with which their ideas are translated into motor actions without any sign of control or inhibitory power.

The motor actions themselves are often so irregular and variable that they have received the name Variable Chorea (Brissaud) or Variable Tic. Sometimes, however, they are limited to one muscle or group of muscles.

In patients who show considerable mental disturbances it is not rare to find fixed ideas and obsessions; also peculiar dislikes and fears, of death, of water, of knives, of animals, etc. With these there is often a condition of melancholia or hypochondriasis.

But besides these peculiar dislikes, these patients often exhibit ridiculous predilections, *e.g.* always to occupy the same seat, to enter a room by a special path, to step on certain stones when walking on a footpath. Others show an exaggerated love of order, an onomatomania, or an arithmomania.

On the affective side also they show the same variability, the same excesses of fear, and the same aversions and attractions. These cases belong generally to that large group of degenerates in whom physical and mental stigmata are correlated; though it must be admitted that some show considerable mental vigour, and it is difficult to discover any mental stigmata.

Frequently patients who exhibit symptoms of tic have, in their childhood, shown marked moral and intellectual instability, which more or less persists, and Meige and Feindel say that such individuals possess a psychic development which belongs to an earlier age than that which they have really reached; that they are in a condition of psychic infantilism.

R. G. Rows.

**CLINICAL AND EXPERIMENTAL OBSERVATIONS ON KATA-**

(411) **TONIA.** LEWIS C. BRUCE and A. M. S. PEEBLES, *Journ. Ment.*

*Sc.*, October 1903.

THIS paper is written from observations on twelve cases of katonias: ten men and two women. Hereditary predisposition was

present in six cases. The disease may be divided into three stages:—

1. Prodromal stage.—Insidious in onset with listlessness and failure of nutrition. Hallucinations of hearing of a distressing nature then appear, leading either to impulsive actions or to paroxysms of fear with complete loss of self-control.

2. Acute stage.—Characterised physically by loss of appetite, rapid and irregular cardiac action, and a rise in arterial blood pressure. Profuse perspiration accompanied the mental paroxysms and pustular rashes were common. Taste and smell were often completely disorganised, and diminished sensibility to touch, heat and pain was present. The skin and tendon reflexes were exaggerated. The pupils were dilated and reacted sluggishly to light. Katatonic spasm occurred at intervals in the voluntary muscles. Sleep was deficient. The temperature tended to be irregular. The habits were faulty.

Mentally, the patients were confused and suffered from vivid auditory hallucinations leading to paroxysms of impulsiveness. Between these paroxysms they might lie for hours apparently oblivious of their surroundings.

The acute stage lasted four weeks, and in half the cases terminated with a distinct febrile attack accompanied by a high leucocytosis, the latter being present in all cases.

A bacterial examination of the blood was made in eight cases during this stage. In one instance a pure culture of a short streptococcus was obtained from the blood of a patient who had passed into the typhoid state before entering the third stage of the disease.

The blood of this patient agglutinated a broth culture of the organism (in a dilution of 1 in 30) in twenty-four hours. The authors tested the agglutinative power of the blood of all the acute and three demented cases to this streptococcus: eight gave definite clumping, three partial reaction, four no reaction. No control ever gave a reaction; and, on testing the agglutinative power of the blood of fifty patients, not cases of katatonia, to this streptococcus, only five gave the agglutinative reaction. It is probable, therefore, that the agglutinin frequently present in the blood of the katatonic is a specific agglutinin.

3. Stuporose stage.—Characterised physically by a disordered alimentary tract, a slow cardiac action with cold extremities, a fall in arterial tension, a liability to pulmonary tuberculosis, a sub-normal temperature, a continued amenorrhœa, and a tendency to retention of urine. The voluntary muscles resisted attempts at passive movement.

Mentally the patients were stuporose, though they were alive to their surroundings and exhibited impulsive actions, curious attitudes, mutisms, etc.



The high leucocytosis, which characterised the termination of the acute stage, fell with the onset of stupor and continued on an average between 12,000 and 16,000 per c.mm.

In three cases which recovered the leucocytosis never fell below 12,000 per c.mm., nor the polymorphonuclear cells below 60 per cent. In four cases exhibiting symptoms of terminating in dementia a low leucocytosis with a low percentage of polymorphonuclear cells was observed.

Some experiments were made on rabbits with a culture of the streptococcus obtained from the blood of the patient while in the acute stage of katatonia. The rabbits were infected through the alimentary tract or blood stream, and a condition of malaise with irregular temperature, increased skin reflexes, and mental hebetude was induced. In healthy rabbits this condition terminated in six weeks, and a condition of immunity to the organism was established. Attempts to pass the organism through an animal and obtain it again from the blood resulted in failure.

A goat was immunised to the streptococcus obtained from the acute case of katatonia. This goat's serum was administered either subcutaneously (12 c.c. daily) or orally (10 c.c. to 140 c.c.) to patients in the stuporose condition with no curative effect.

Five stuporose cases treated with subcutaneous injections of broth cultures of the organism killed by heat showed no material mental improvement; on the other hand, the same procedure aborted the onset of acute exacerbations of the disease in one patient during the acute stage of his affection: the injections (beginning with 4 c.c.) being given on the appearance of the prodromal symptoms of an attack.

As it was impossible for an immune body to be formed in the short period (two to three hours) which elapsed between the injection and the improvement in the patient's condition, the beneficial effect produced in this case cannot be explained by any theory at present held with regard to the production of immunity.

H. DE M. ALEXANDER.

**TWELVE CASES OF "KORSAKOW'S DISEASE" IN WOMEN.**  
(412) JOHN TURNER, *Journ. Ment. Sc.*, October 1903.

SOUKANOFF and Boutenko have analysed the total number (192) of cases collected: 112 occurred in men and 80 in women, and three-fourths of the cases were of alcoholic origin. Complete recovery was very rare in the male cases.

All the author's cases presented the symptoms described by Korsakow, viz., amnesia, disorientation, pseudo-reminiscence, and confabulation. An alcoholic history was present in ten, and

highly probable in the other two. Ten of the patients were unable to walk or stand, one had unsteady gait, and in one the gait was normal.

Peripheral neuritis occurred in ten, probably in all; the exaggerated knee-jerks present in some of the cases did not forbid this assumption, as the author found the posterior tibial nerves degenerated in a woman who had died with symptoms of peripheral neuritis associated with very exaggerated knee-jerks.

The speech presented nothing characteristic. Examination of the eyes revealed unequal pupils in three cases, normal reaction to light and accommodation in six, and diminished reaction to light in the remainder. Four cases presented symptoms of delirium tremens at some period of their affection, two of them having typical visual hallucinations (rats and other animals). Seven cases were discharged; in one of these the memory regained its normal state. Three remain. Two died: one after a month, the other after four years.

The author agrees with Jolly that the symptoms in this affection are *one* of the manifestations of the action of alcohol and other toxins on the nervous system, and believes that the specific action of these toxins is on the nerve-fibres and not on the nerve-cell. Peripheral neuritis occurs if the peripheral fibres are implicated; if, on the other hand, the toxine expends itself on the prolongations of the cortical cells passing down the cord in the pyramidal tract, or on those which pass up the cord in the posterior columns, a central neuritis ensues.

The nerve-cell change met with takes the form known as axonal reaction; and it can be accounted for without suggesting any direct toxic action on the cell bodies, as it can be produced by severance of axons from their cells, or by influences which injuriously affect the axons (*e.g.* hæmorrhage). Deprivation of those normal stimuli which pass to the efferent cells (stimuli proceeding from sensory cells and impinging on motor cells) is also capable of inducing this change; this factor is present in Korsakow's disease, and in every case where a microscopical examination of the cord has been made, degeneration of the posterior columns has been found.

Probably in all cases there is implication of the association nerve-fibres, especially those of the tangential system; and while this factor may be held accountable for the interference in the memory and the time and space ideas, it is probable that the polyneuritis also assists in fostering the peculiar mental troubles.

Consciousness depends upon the integrity of the periphery: a case is on record of a deaf man with practically total anæsthesia of the skin, in whom simply closure of his eyes was sufficient to make him sleep, *i.e.* become unconscious.

Our time and space perceptions depend upon the appreciation of the sequence or simultaneity of impressions. If these are interfered with or perverted, then we get perversions of time and space ideas; if there is widespread abolition of these impressions, then not only will the subject be disoriented but his ideas of present time and space will be annulled; and, not receiving an adequate supply of sensations from the periphery, he will draw on the ideas already stored up, thus producing pseudo-reminiscence and confabulation.

In Korsakow's disease the peripheral trouble only assists in forming the psychic symptoms, as the confusion of mind is due to disorder of the central nervous system; and patients, who have recovered from the peripheral defects, often still present psychical disturbances, *e.g.* amnesia and pseudo-reminiscence.

H. DE M. ALEXANDER.

**ON THE AGE OF ONSET AND THE INFLUENCE OF HEREDITY  
(413) IN THE PATHOGENESIS OF DEMENTIA PRIMITIVA  
OR PRÆCOX.** MARCO LEVI-BIANCHINI, *Riv. speriment. di  
Freniatria*, 1903, f. 3, p. 558.

IN 125 cases of his own and 105 observations collected from the literature of the subject, the author found that 13 was the lowest age and 56 the highest for the onset of dementia præcox. In 53 per cent. the disease began before the age of 25. With regard to special forms, 56 per cent. were hebephrenic, 36 per cent. paranoid, and only 1·8 per cent. catatonic cases. Hebephrenia is consequently the most important form; a genuine and permanent catatonia occurs very seldom, if at any time, without passing over, earlier or later, to a hebephrenia or a paranoid dementia, and the author would therefore be inclined to recognise only these two forms. His own cases constituted 27 per cent. of the number of inmates, both sexes contributing about equally. Hebephrenia occurred more frequently in women, paranoid forms in men. Believing that Kraepelin, who sets down the proportion of cases of paranoid dementia as 40 per cent., under this head has entered many cases of hebephrenia, the author maintains that the name dementia paranoides should be reserved for cases in which the delusions remain constantly as a stereotypy of delirious conceptions.

(The dividing of the group dementia præcox is at present mostly a matter of convenience, no distinct frontiers existing between the traditional three forms. As the author does not give a statement of his principles of distinction, and as his statistical data show that his point of view is not quite the same as that of Kraepelin to whom he refers, it is scarcely possible to make any

use of these statistics relating to special forms for comparison with those of other authors.—*Rep.*)

Heredity was found in 59 per cent.; if only mental diseases are reckoned, in about 40 per cent. In that respect the forms presented no difference worth mentioning.

The mental faculties, prior to the onset of the disease, were normal in 90 per cent. (average, 60 per cent.; below average, 24.4 per cent.; above average, 6 per cent.). In the rest a feeble-mindedness was recognised; in one case hysteria, in another epilepsy were already present. Over-average mental faculties were never found to have been possessed by those who developed catatonia.

HANS EVENSEN.

**THE CRIMINAL; AND THE FUNDAMENTAL ANOMALIES OF  
(414) CHARACTER.** F. DEL GRECO, *Il Manicomio*, Vol. xix., No. 2.  
1903.

IN this article, the continuation of a preceding study of the biological factors of criminal individuality, Prof. Del Greco compares the anomalies of character observed in delinquents, with similar or analogous manifestations in degenerates and normal individuals, and by this comparison attempts to demonstrate their common factors, mode of origin and development.

He clears the ground for investigation by excluding from his examination the criminal by passion and the occasional criminal, and restricts this to habitual and born criminals. Between these two a sharp line of distinction is drawn. The former are weak and passive, suggestible and easily susceptible of evil influences, and fall into crime under slight stress of circumstance. When in association they are obedient and plastic to their company, and are the beasts of burden, the fatal executors and necessary accomplices of the born criminal.

Anti-social from lack of initiative and moral personality, they are indolent rather than inherently wicked.

The born criminal, on the other hand, is above all anti-social. He may be of weak mind, and though frequently insensitive to pain is timorous on occasion; he is one-sided in his affections and dominated by a great fund of organico-instinctive impulse. He is dead to remorse and commonly possessed of a notable mobility of temper and expression.

The author agrees with Ferri that a fundamental characteristic of the born criminal is an imperfect social sense, depending on a deep-seated distrust of other men. This is not acquired but original, though possibly accentuated by the attitude to them of others. This distrust or suspiciousness it is which is the chief index to a degeneracy of character which is to be found in all born criminals, and in many morbid states, notably that of paranoia.

In the normal man, character results from the operation of opposing forces, defensive and repulsive on the one hand, sympathetic and assimilative on the other.

In the paranoiac and criminal, the former preponderates over the latter, have their base in the arrest of every sympathetic activity, and if they do not end in impulsive aggressiveness, certainly negate any social and humane activity.

The social life is, the author affirms, the matrix of character, and in tracing the phylogenetic evolution of man and the origin of society with its implicate and obligatory duties, he shows that with the ascending development of character there is an increasing subordination of personal, biological, organico-psychical elements, to socio-psychological elements, joint interests, or states of solidarity.

In the degenerate, the paranoiac and the criminal, there is a reversal of this process, a return to a more primitive attitude, depending on an intrinsic restriction of the personality, actual and potential. This restriction produces a profound dissonance with surrounding social conditions, which finds expression in a distrust or suspiciousness of others, revealed in behaviour and an isolated and recalcitrant disposition. In the paranoiac this is evidenced by stable delusions of persecution; in the habitual criminal, by passivity, incoherence of action, and suggestibility to evil; and in the born criminal by an organic impulsiveness, egoistic, anti-social, anti-humane.

R. CUNYNGHAM BROWN.

**REGICIDES ; SANE AND INSANE.** By E. C. SPITZKA, *N. Y. Med.* (415) *Journ.*, Aug. 15th to Sept. 5th, 1903.

DR SPITZKA, well known as a critic of Lombroso's teaching, discusses in this paper the evidence he has collected as to the character, and in so far as it was obtainable, the mental condition of the actors in a large number of magnicides. Armed with these facts he attacks the definition of the type of regicide as formulated by Dr Regis. This definition runs as follows:—

“Regicides are degenerates of a mystic temperament, who, led by a political or religious delusion, at times complicated by hallucinations, think themselves called on to act the double rôle of judiciary and martyr; and under the influence of an obsession that is irresistible, kill some great personage in the name of God, the country, liberty or anarchy.”

Collateral propositions state: First, that the regicide is “by his very nature a *solitaire*, having ordinarily neither accomplices nor confidants, even amongst his nearest intimates.” Second, that regicides are “sick persons fanaticised to the point of delusion,” and so alike that they correspond “trait for trait.” Third, that

a noteworthy feature is the exceptionally juvenile age of the regicide.

Dr Spitzka's investigations cover a large field, and the practical result is this : that the past of the regicide, in which, according to Regis' definition, the martyr purpose develops on a basis of degeneracy, fails to be sustained in 43 per cent. of the cases; that the *solitaire* doctrine breaks down in 93 per cent. ; and that the dementia into which, according to the author, they should fall, lacks verification in 97 per cent. of those who have escaped the extreme penalty of the law.

Dr Spitzka does not deny that in the case of magnicides and political assassins generally, a high ratio of demonstrably insane actors exists ; indeed, so far as his own experience goes, he would predicate insanity in the majority of magnicides ; but he contends that this ratio is not sufficiently high to justify any *a priori* presumption. Further, he says, traditional cases show that the very regicides whose insanity is admitted on all sides, are more remote from the standard definition as formulated above, than those alleged to be unsound, but whose mental integrity has never before been impeached. With regard to the *solitaire* question, Dr Spitzka says that one of the most trivial discriminations is that between singleness and partnership in knowledge of an assassin's plans. The ratio of *societaires* to solo assassins is practically as 2 to 1, and this alone should dispose of the question whether the regicide is "by his very nature" a *solitaire* or not. Throughout this interesting paper Dr Spitzka's attitude is polemical rather than judicial, but the figures he gives, even though they rest on evidence often conflicting and uncertain, and necessarily not at first hand, go to prove that Regis' definition is another instance of premature formalisation and is not of general application.

R. CUNYNGHAM BROWN.

## TREATMENT.

**SUBCUTANEOUS DRAINAGE IN THE SURGICAL TREATMENT (416) OF HYDROCEPHALUS INTERNUS.** NICHOLAS SENN, *Alienist and Neurologist*, August 1903.

DR SENN divides his paper into three parts: (1) Introduction; (2) What has so far been accomplished by Puncture and Drainage of the Ventricles; and (3) A Case of Hydrocephalus Internus, complicated by Spina Bifida. Subcutaneous Drainage of the right Lateral Ventricle. Elastic Compression of Skull.

In the introduction the author says the utter futility of all kinds of internal and external medication in the treatment of hydrops of the ventricles of the brain is generally conceded. Post-mortem findings tend to prove that this condition results most

frequently from meningeal tuberculosis, but there can be no question concerning the occurrence of hydrocephalus independently of tuberculosis in the form of a simple transudation or uncomplicated hydrops, and it is in this class of cases that he looks hopefully to some sort of successful intervention.

With regard to puncture and intraventricular medication, he says it is very seldom the case that puncture and evacuation of hydrocele in the adult results in anything better than temporary relief: a recurrence, sooner or later, is the rule. If this is the outcome of a very large experience with this affection, it is not surprising that the results of the same treatment of hydrops of the ventricles under greatly more complicated conditions should have been no more successful. The temporary improvement following this treatment is always of short duration. In only one of Dr Senn's cases did this treatment succeed. The patient was a young man, the subject of vague cerebral symptoms which pointed to abnormal intracranial pressure. He trephined over the middle of the fissure of Rolando. The needle of an exploring syringe was inserted in the lateral ventricle and about two ounces of clear cerebro-spinal fluid escaped. An ounce of a weak aqueous solution of iodine was injected. The operation was followed by a marked cerebral irritation which subsided after a few days. The improvement which followed was a decided one and proved to be of a permanent character. This is an isolated case, so that complete drainage of the ventricles was resorted to. There are two great risks with external drainage: (1) Too rapid evacuation of the cerebro-spinal fluid and its consequent harmful intracranial congestion; (2) Infection. Dr Senn endeavoured to get over these difficulties by his subcutaneous method of drainage. The case referred to is that of a child who had been operated on for Spina Bifida when it was two days old. On the day after the operation the head began to enlarge, and had continued to do so till the child came under the care of Dr Senn, when it was ten months old: the head then measured twenty-six inches on a level with the frontal eminences and occipital protuberance and twenty-nine inches from ear to ear. There was no ossification of any of the sutures, all the cranial bones were widely separated and freely movable and the fontanelles were large and pulseless. A horse shoe shaped incision was made down on to the bone over the coronal suture on a level with the frontal eminence on the right side. The ventricle was punctured with a grooved director, a small rubber drainage tube was then inserted along the groove and the remaining portion of the tube was inserted into a large pocket in the temporal region. The skin flap was then stitched and the head was dressed in absorbent cotton and a very thin rubber bandage applied. No anæsthetic was used. The circumference of the head gradually

diminished until the patient's death, a week after operation, when it measured twenty-four inches. The temperature during the week ranged from 102-104° F. and the pulse was rapid. The drainage tube was removed the day before the child died, when it was found to contain clear cerebro-spinal fluid, and the lumen of the tube was patent throughout and free from fibrinous masses.

Dr Senn sums up: "Unfortunately no post-mortem was made, being objected to by the parents. The immediate cause of death is obscure to me. The pulse and temperature would suggest infection; but all the usual brain symptoms so constantly attending acute meningitis were absent. The swelling outside of the skull was caused solely by the cerebro-spinal fluid. At the time the tube was removed this fluid was as clear as when the ventricle was first opened. The rise in the temperature set in a few hours after the fluid was drained into the subcutaneous connective tissue, and the question might arise whether or not by the absorption of this fluid some phlogistic substance found its way into the general circulation. Although this case terminated fatally, as most of such cases will under any kind of treatment, it is apparent that subcutaneous drainage is preferable to open drainage for reasons that have already been enumerated. This case proves conclusively that the cerebro-spinal fluid is quickly absorbed by the connective tissue, almost as soon as it escapes."

R. G. WHITE.

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## Review

**CATATONIA AND STUPOR.** Dr A. CLAUS, Brussels. Seveyrens, 1903.

THIS monograph was presented at the Congress of Alienists and Neurologists of France and Belgium held at Brussels in August 1903. It is not so much an original investigation into the subject of catatonia as an epitome of the views of various authors on this much-discussed question. It shows very clearly the great divergence of opinion that still exists, even among those very well qualified to judge. The disease was first described as an entity nearly thirty years ago (1874) by Kahlbaum, and though it has, especially during the latter half of that time, been the subject of very vigorous discussion, one sees no signs of even moderate unanimity of opinion regarding it. In this respect, however, it does not differ from the larger subject, of which indeed it is merely a branch, the classification of mental disease.

The first part of the book is devoted to a general review of the whole subject in its historical aspect. This is considered necessary in order to show the conceptions of mental disease

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which were prevalent when Kahlbaum published his description of catatonia, and to indicate the steps which led up to it. The views of the French and German schools of alienists are briefly summarised. Particular attention is given to hebephrenia, as it has a special relation to catatonia. It is stated that Kahlbaum, in elevating catatonia to the dignity of a distinct disease, was governed by a desire to draw a parallel between it and general paralysis. He therefore laid special stress on the bodily symptoms, and gave the disease the alternative name of *Spannungs Irresein*. An account is then given of the symptoms of catatonia, of its usual four stages, and of the characters of each of these. The most characteristic stage is the third, in which there usually exists more or less of the spasticity (*Spannung*) which gives the alternative name to the disease. He admits, however, that certain stages may be absent and that the typical picture may be departed from. Kahlbaum's paper remained almost unnoticed for some years. This is accounted for by the warm discussion initiated by Westphal in 1896 on the acute forms of insanity. This discussion is rather elaborately analysed by Dr Claus, because in the end the question of catatonia became involved. He also gives a short account of the views of French alienists of that time on the same subject.

In the second chapter the author gives the opinions which have been expressed by alienists on the subject of catatonia. These are divided into three classes. By some catatonia is regarded as a morbid entity, by others as a morbid syndrome, while others again look on it as a form of *dementia præcox*. In the first group are found Hecker, Jensen, Hammond, Spitzka, Neisser and Ziehen; in the second, Westphal, Mendel, Sander, Séglas, Chaslin, and others; while in the third the chief name is that of Kræpelin. The views of each of these authors are given in summary form, especially those of Kræpelin. His views are traced through the various editions of his "Text-book of Mental Diseases," and are seen to have undergone very considerable modification. In his last edition cases of catatonia are classed, along with hebephrenia and some cases of paranoia, under the heading of *dementia præcox*. Kræpelin's views have met with acceptance by many in Germany, and appear to be gaining more numerous adherents there and elsewhere.

In the next chapter the etiology and pathogenesis of catatonia are considered. As regards age, the majority of writers conclude that more than 50 per cent. of the cases occur before the age of twenty-five. Sex appears to exercise little influence. Heredity plays a varying part according to various authors, but it is observed that the more carefully the history is gone into the higher is the percentage in which hereditary defect is found. Kræpelin places it as high as 70 per cent., Ziehen at 80. The

influence of intoxications, infections, and traumatisms is referred to.

In the last chapter the symptomatology of catatonia is discussed. On account of its close relationships to this disease, stupor is first of all analysed. It is a prominent symptom in catatonia and is found in several other affections. The views of Mendel, Ziehen, Chaslin, Meynert, Dufour, Newington, and others on this subject are given. Attention is then turned to the physical symptoms of catatonia. Kahlbaum, as has been mentioned, attaches much importance to these. They vary much in different cases, and for this reason do not have nearly the same weight as do the physical symptoms in general paralysis. In catatonia they seem to depend much more intimately on the mental state and the degree of mental reduction, and cannot be regarded as indicative of associated disease of the motor centres. In closing, reference is made to the pathology of the disease and various views are stated. But they are more or less discordant, and at the present time one must confess almost complete ignorance of this difficult matter.

On the whole the book is somewhat disappointing. It is a summary of opinions and does little to forward the problem which Kahlbaum professed to have solved, but which many still believe to be unanswered. It, however, gives a useful review of what has been done in this subject.

JAS. MIDDLEMASS.

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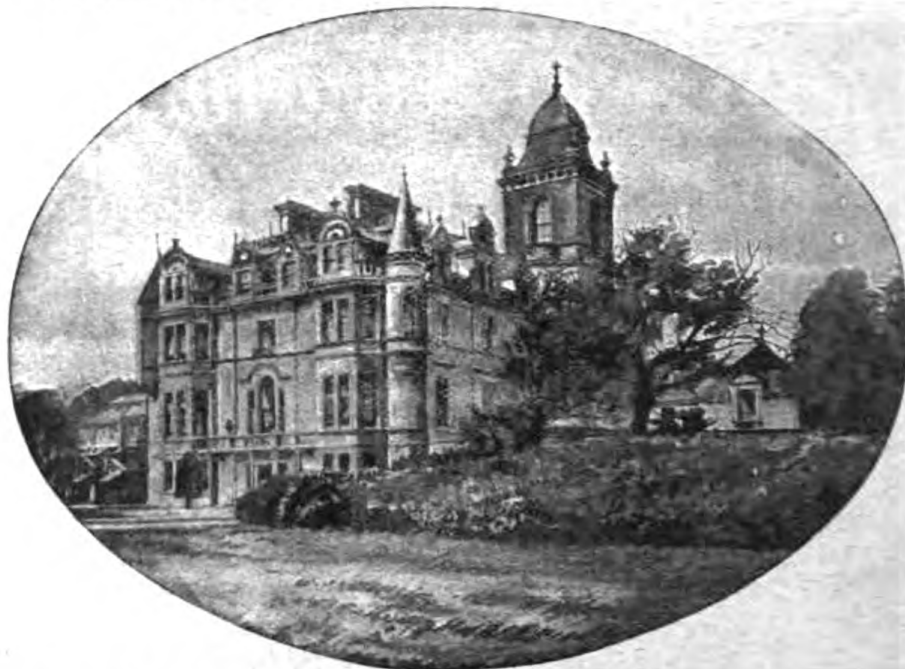
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